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PLANT DISEASES

BY

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PREFACE

ALTHOUGH the subject of Plant Pathology has assumed great scientific and practical importance, there has been no general, up-to-date account of the diseases of British crop plants available for some years. The present book endeavours to supply this deficiency. In it the author has also attempted to include an outline of our present knowledge of important plant diseases in other parts of the Empire and in other countries of the world. For a considerable period Massee's *Diseases of Cultivated Plants and Trees* has been the standard work on this subject in Britain, but the last edition was published in 1915, and since then Mr. Massee has died.

The author has had a good deal of experience in training men to serve as Plant Pathologists at home and abroad, and his book is designed particularly to assist such students and others who are carrying out investigations on Plant Diseases. It is hoped also that the book will be useful to the general botanist, to students of Agriculture, Horticulture, and Forestry, and to those cultivators of the soil who take an enlightened interest in the crops they grow.

Extensive use has been made of existing books on Plant Diseases, and the author gratefully acknowledges his indebtedness to the following, among others: Marshall Ward's *Disease in Plants*, Butler's *Fungi and Disease in Plants*, Petch's *Diseases and Pests of the Rubber Tree*, and *The Diseases of the Tea Bush*, Nowell's *Diseases of Crop Plants in the Lesser Antilles*, Cunningham's *Fungous Diseases of Fruit Trees in New Zealand*, Bewley's *Diseases of Glasshouse Plants*, Hiley's *Fungal Diseases of the Larch*, Grove's *British Rust Fungi*, Heald's *Textbook of Plant Diseases*, Duggar's *Fungous Diseases of Plants*, Smith's *Bacteria in relation to Plant Diseases*, Chupp's *Manual of Vegetable Garden Diseases*, Stevens's *Plant Disease Fungi*, Hartig's *Diseases of Trees*, Prillieux and Delacroix's *Maladies des Plantes Agricoles*, and Sorauer's *Pflanzenkrankheiten*. *The Report on the Occurrence of Fungus*,

Bacterial, and Allied Diseases of Crops in England and Wales for 1922-4, compiled by Dr. G. H. Pethybridge and published by the Ministry of Agriculture and Fisheries, has been particularly useful in dealing with British plant diseases. For most of the fungi the classification used in Gwynne-Vaughan and Barnes' book, *The Structure and Development of the Fungi*, has been adopted chiefly, but in the higher Basidiomycetes the scheme employed in Rea's *British Basidiomycetae* has been followed.

The author is under deep obligation to a large number of friends for information and valued criticism, especially to Dr. G. H. Pethybridge, Dr. W. J. Dowson, Mr. W. C. Moore, Dr. E. J. Butler, F.R.S., Mr. J. Ramsbottom, Prof. Sir Rowland Biffen, F.R.S., Prof. E. S. Salmon, Mr. T. Petch, Mr. A. Sharples, Mr. C. G. Hansford, Dr. H. H. Storey, Dr. A. Thaysen, Dr. R. M. Nattrass, Mr. F. L. Engledow, Miss E. Wakefield, Dr. I. Hoggan, Miss K. Sampson, Mr. S. P. Wiltshire, Dr. Malcolm Wilson, Dr. H. Wormald, Mr. W. E. Hiley, Prof. N. J. G. Smith, Mr. L. Ogilvie, Mr. R. W. Marsh, Mr. R. V. Harris, Dr. A. Smith, and Mr. R. G. Tomkins. To Dr. W. J. Dowson the author is under a particular debt of gratitude for reading the whole of the proofs.

All the illustrations are new, and to Dr. W. J. Dowson special thanks are tendered for his great kindness in making a large number of them. Other illustrations have been very kindly provided by Dr. G. H. Pethybridge, Dr. R. M. Nattrass, Dr. Somerville Hastings, Mr. G. O. Searle, Dr. Kenneth Smith, Mr. R. W. Marsh, Dr. A. Smith, and Mr. C. R. Metcalfe.

The common names assigned to the diseases of British crops are those which have been compiled by the Plant Pathology Sub-committee of the British Mycological Society. References to literature will be found at the end of each chapter.

The book is dedicated with deep respect to the memory of Professor H. Marshall Ward, who first stimulated the author to take up the study of Plant Diseases.

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CONTENTS

CHAPTER I

INTRODUCTION

By disease in plants is meant some disturbance in the normal life-processes which affects either a particular organ or the entire plant, and which sometimes leads to premature death. Cultivated plants are usually more liable to disease than wild plants, partly because, under cultivation, large numbers of the same kind of plant are grown together, so that disease spreads rapidly if established, and partly because many crop plants, selected and preserved to provide man with food and materials, are constitutionally very susceptible to disease and would not survive the stress of competition in nature. Furthermore, plants are often cultivated under conditions vastly different from those under which they or their wild progenitors grow in nature, and though by careful husbandry they may thrive under these artificial conditions, the danger of disease is always considerable. In nature, a balance has been struck between the different kinds of plants and animals inhabiting a certain area, but in crop cultivation the balance of nature is constantly disturbed.

Losses caused by plant diseases

The losses caused by plant diseases are sometimes enormous, and the cultivation of certain crops has had to be abandoned in some countries owing to the ravages of disease. At the present time 'Panama disease' threatens to destroy the cultivation of bananas in Central America and the West Indies, and the growing of limes in Montserrat may have to be abandoned owing to 'Withertip' disease. One of the causes which led to the cessation of coffee cultivation in Ceylon in the latter half of the nineteenth century was the increasing prevalence of the rust fungus, *Hemileia vastatrix*.

Losses through disease in storage are often severe. In 1921–2 certain cargoes of apples shipped in sound condition

from Australia were found to be almost worthless on arrival in England on account of disease. The wastage of potatoes and roots stored in clamps during the winter is sometimes enormous.

The toll exacted by certain diseases is difficult to estimate, for no very marked effects are produced on the plants. English wheat attacked by Yellow Rust (*Puccinia glumarum*) is hardly affected in its growth, yet it is certain that this fungus causes an insidious drain upon the crop, leading to a loss of from 5 to 10 per cent., largely owing to shrivelling of the grain.

With some diseases, such as Common Scab of potatoes, the loss is occasioned chiefly through unsightliness, which detracts from market value.

The causes of plant diseases

Disease in plants may be brought about either through attack by some kind of parasite or by some autonomous, functional derangement. Many non-parasitic diseases have not been closely investigated yet, and in some respects these diseases are more difficult to study than are those caused by parasites. Abnormal water relations, peculiarities of soil, extremes of temperature, and interference with respiration are a few of the many causes of functional disturbances in plants.

In recent years parasitic plant diseases have been studied very intensively, and it is now known that many different groups of organisms attack plants parasitically.

It has recently been shown that certain Protozoa, allied to the trypanosomes that cause disease in man and animals, live sometimes in the laticiferous tissues of plants, producing symptoms of ill-health, but so far as is known no serious crop disease is due to these organisms. Nematode worms of microscopic size often invade plants, and, living parasitically therein, cause serious disease in roots, tubers, bulbs, stems, and leaves, but it is beyond the scope of this book to deal with this group of parasites.

Highly infectious diseases of the 'virus' type are now recognized to be some of the most serious that affect plants.

Much remains to be discovered about the etiology of this group, but it is sometimes considered that these diseases are caused by organisms of ultramicroscopic size. Bacteria, long known as some of the most important pathogenic agents in human disease, have also been shown to be responsible for many important plant maladies. The curious group of the Myxomycetes also contains a few serious parasites.

The Fungi include an immense number of forms, parasitic on plants, which are often extremely injurious. It was, indeed, in connexion with the study of fungus diseases of plants that the science of Plant Pathology was founded by Berkeley, de Bary, and others.

Certain Green Algae invade the tissues of crop plants and cause serious disease, as in ' Red Rust ' of tea.

Invasion of the higher plants by micro-organisms does not, however, invariably cause disease. Many examples are now known in which the bacterium or fungus not only thrives within the flowering plant without causing injury, but actually confers benefit upon it, as in the bacterial nodules of leguminous plants and the mycorrhizal associations between fungi and flowering plants. As between the higher plants and associated micro-organisms every relationship may be exhibited, from the most destructive type of parasitism to a completely harmonious symbiosis.

Parasitic flowering plants are also sometimes destructive to crop plants, as e. g. Dodder (*Cuscuta Trifolii*, Bab.) on clover. In the rubber plantations of the eastern tropics, mistletoes (*Loranthus* spp.) may weaken the trees considerably.

Symptoms of plant diseases

The symptoms exhibited by plant diseases are extremely varied. These effects are fully described in the latter part of this book, but some of the commoner manifestations of disease may be mentioned briefly here. The entire appearance of the plant may be transformed, as in certain virus diseases, or parts only of the shoot system may be greatly modified, as in the ' witches' brooms ' of trees, caused by fungi. Invasion of the woody tissues of the stem by micro-organisms often leads to

'die-back' of the aerial parts.   Fungus attack of the root
system is frequently followed by sudden death of the plant
without premonitory symptoms, although wilting or loss of
foliage may precede death.   Invasion of the bark, accompanied
by only a limited extension of the parasite, often produces
'cankers'.   Parasitic attack of seedling plants at soil level
often causes 'damping off'.   The development of spots and
blotches on leaves and young stems is usually due to micro-
organisms, and such necrotic areas may be so numerous as
to lead to death of the entire shoot system.   Rotting of the
softer parts of shoots and fruits may be caused by a variety
of organisms.   Particular organs of the host plant are some-
times transformed by the reproductive parts of the parasite,
as in the diseases of cereals caused by Smut fungi.   Lastly,
the parasite may stimulate the invaded part of the plant to
grow extravagantly, so that large swellings or galls are formed,
as is seen in Wart Disease of potatoes and in Crown Gall.

   The above types of symptoms are mostly shown in tissues
actually occupied by the parasite, but signs of ill-health may
be induced in organs remote from the seat of the parasite, as
in certain 'wilts' and in the early stages of Silver-leaf disease
in fruit trees.   This 'action at a distance' is brought about by
the secretion of toxic substances by the parasite, which are
carried away by the sap-stream into remote regions, where the
symptoms of disease often first appear.

Dissemination of plant diseases

Wind is one of the most potent factors in the dispersal of
parasitic fungi, their spores being readily carried in a living
condition by air currents for considerable distances.   The
ability of wind to distribute spores over wide tracts of land
and sea has, however, probably been over-estimated in the
past.

   Movements of water in the soil may disperse parasites
which attack underground organs, and some of these organisms
have a limited power of free movement in water when in the
reproductive condition.

Insects often disseminate the germs of disease, and many virus diseases appear to be spread exclusively by their agency. In feeding on diseased tissues the insect becomes contaminated by the infectious principle, and when it subsequently feeds upon a healthy plant the virus is transmitted. Insects also often carry spores on the surface of their bodies.

The dissemination of diseases of crop plants has been greatly facilitated at times by the operations of man himself; many diseases are now known to have been introduced into new countries by the importation of parts of plants, especially seeds, tubers, and bulbs, in an infected condition or bearing spores upon their surface. The germs of disease are often distributed by the careless transfer of contaminated plant receptacles from one district to another, and by their inclusion in particles of soil adherent to tools, boots, or the feet of animals.

Epidemic and sporadic plant diseases

The diseases most to be feared are those which are epidemic in character, i. e. those which develop almost simultaneously and universally throughout a crop. For a parasitic disease to become epidemic an abundant supply of infecting units must be available, the host must be in a susceptible state, and the conditions of weather or soil, or both of these, must be favourable for infection. Sporadic diseases, on the other hand, merely affect a plant here and there, although if neglected, so that the germs of infection multiply unduly, a disease of this kind may tend to become epidemic, as occurs when Silver-leaf disease of fruit trees remains unchecked.

Susceptibility and resistance to disease *

Nearly all cultivated plants exist in innumerable forms or varieties, which often differ markedly in susceptibility to disease under identical environmental conditions. The degree

* For further discussion of the literature on this subject see Butler, E. J., *Fungi and Disease in Plants*, Calcutta, 1918; Blackman, V. H., *Physiological Aspects of Parasitism*, Presidential Address, Section K, British Association for the Advancement of Science, Toronto, 1924; Brooks, F. T., 'Disease Resistance in Plants', *New Phytologist*, vol. 27, p. 85, 1928.

of susceptibility or resistance of a particular variety to a certain disease is a definite genetic character known to be transmitted hereditarily in many cases according to Mendel's law. Although resistance to disease is always, in the last analysis, a property of the host protoplasm, it is often possible to point to some special feature in the organization or metabolism of the plant with which the resistance is intimately associated. A slightly thicker cuticle may prevent the leaves of one variety from being penetrated by fungus germ-tubes, the rapid formation of cork or gum barriers may preclude a fungus from spreading extensively after infection has been initiated, or a too vigorous onslaught by the parasite—as in some Rust fungi—may inhibit the establishment of a life together between host and parasite.

If the invaded tissues are not killed forthwith by the parasite, the host and parasite react on one another. Antibodies may conceivably be produced by living host cells in response to parasitic attack, but little is known about these substances in plants, and doubt is sometimes expressed whether they occur at all in them. Recovery of a plant from disease is sometimes due to progress of the parasite being stopped by the formation of cork or gum barriers in the tissues, with the result that the invading organism dies out. Recovery from a disease does not confer immunity from subsequent attack, as in certain human diseases.* In this connexion it must be borne in mind that the circulatory system of plants is vastly different from, and far inferior to, that of the higher animals, and that many plants are constantly forming new parts to which any antibodies that may conceivably have been formed in the older tissues could only be transmitted with great difficulty.

A crop plant which is severely attacked by one disease may thereby be rendered unusually susceptible to another disease. For example, 'Little Joss' wheat, when attacked by Bunt

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* Dr. W. J. Dowson informs the writer that in Kenya young coffee plantations, after being once vigorously attacked by *Hemileia vastatrix*, are not again so virulently affected. It seems that in this case a certain degree of resistance is acquired.

(*Tilletia caries*), is quite susceptible to Yellow Rust (*Puccinia glumarum*), although it is generally resistant to this fungus.

The influence of environment upon the incidence of plant diseases

Although susceptibility and resistance to specific diseases are inherent qualities, the degree of susceptibility or resistance is usually modifiable within considerable limits by variations in environmental conditions. Exceptionally, the immunity from Wart Disease exhibited by some varieties of potatoes appears to be complete under all conditions as far as is known at present, although it has been shown recently in the laboratory that certain varieties formerly thought to be 'immune' as the result of field tests are, in fact, somewhat susceptible.* In general, environmental conditions determine whether disease will become established or not in a susceptible plant when germs are available for infection. With parasites which infect subterranean organs the moisture content of the soil may be too low or the temperature too high to allow of infection. Thus any one factor in the environment may determine whether disease develops or not. The direct influence of the environment on the host must also be taken into account, for the host may be modified in such a way as to render it resistant to infection : thus in hot, dry weather the cuticle may be so thickened as to be impenetrable by fungus germ-tubes.

Weather generally plays the dominant role in the environment as regards the incidence of plant diseases. The most important factors in weather are temperature, sunshine, rainfall, relative humidity of the air, and wind, and any one of these may exercise a dominant influence on infection. The effect of temperature upon the occurrence of certain plant diseases is very important. Black Rust of wheat (*Puccinia graminis*) flourishes at comparatively high temperatures, whereas Yellow Rust (*Puccinia glumarum*) thrives best at

* See page 69.

relatively low temperatures; to a great extent the distribution of these diseases both seasonally and regionally can be correlated with temperature. Rainfall and the deposition of dew also exercise a potent influence upon the establishment of disease in plants, for, as regards infection of flowers, leaves, and young stems, the danger of fungus germ-tubes being killed by desiccation is considerable. Wet weather, therefore, generally favours the development of fungus diseases. With certain mildews, however, moderately dry weather facilitates their development. Both temperature and rainfall also affect soil conditions.

Other physical or chemical characters of the soil may also decide whether disease will occur in the underground parts; for example, Finger-and-Toe disease of turnips occurs only in acid soils. Excessive nitrogen in the soil tends to increase susceptibility to some diseases, while abundant potash enhances resistance. The nature of the soil often determines the depth of the root system, and this in turn influences susceptibility to certain diseases. For instance, on shallow, light soils overlying chalk in England, wheat is less susceptible to Yellow Rust (*Puccinia glumarum*) than on deep, heavy soils; this is related to a deeper root range and a greater nitrogen-intake in the latter. The type of stock on which fruit trees are grafted or budded sometimes exercises a marked influence upon susceptibility to disease.

The control of plant diseases

Plant diseases become established in so insidious a manner that they are often well developed before they can be detected. By the time disease is evident in a plant it is rarely possible to cure it. In comparison with the human pathologist the plant doctor is handicapped by the absence of anything in plants which corresponds to the living blood-stream in man, and by the difficulty of introducing substances to circulate in plant tissues which will be toxic to the parasite but harmless to the host. The efforts of plant pathologists are centred therefore upon the prevention of disease rather than its cure.

The use of disease-resistant varieties is one of the most effective ways of reducing disease in crop plants. It often happens that a variety markedly resistant to a certain disease is undesirable commercially because of low yielding capacity or some other defect. With the knowledge of the laws of heredity now available, the quality of resistance can often be combined with other good characters by the plant-breeder. Geneticists throughout the world are endeavouring to introduce into cultivation new varieties, synthesized by them, which will be more resistant to disease than those formerly grown, and which at the same time will possess the desirable commercial qualities of the older types. New varieties should be tested for several seasons as regards the influence of weather and soil upon their reaction to disease before they are distributed to cultivators. The problem of building up resistant varieties of crop plants must be faced in each country because of the influence of varying environments upon disease and because distinct strains of parasites may occur in different regions. Even in a small country like England, variations of soil and weather necessitate a wide range of varieties of any one crop plant to suit the various local conditions. Plant-breeders use a considerable range of characters in trying to obtain varieties that will not be attacked by disease. A variety which matures a few days before another may be apparently more resistant to disease or may escape attack altogether because spores are not then abundantly available for infection, or because the tissues, being older, are less susceptible.

The adoption of sanitary measures is effective in preventing certain plant diseases.* Plant sanitation or plant hygiene concerns particularly the destruction of diseased material so that the sources of infection are eradicated as far as possible. The most effective way of destroying diseased plants is by fire, failing which, they can be rendered innocuous by chemicals. The methods of plant sanitation are especially efficacious in intensive cultivations such as that of fruit in temperate regions and rubber in the tropics. With Silver-leaf

* Brooks, F. T., ' Plant Sanitation in Fruit Plantations', *Trans. Brit. Myc. Soc.*, vol. 6, p. 253, 1920.

disease, prevention of the causative fungus from fructifying in and upon the confines of fruit plantations tends to reduce infection. Probably no greater care is taken to prevent and control disease in any type of cultivation than in the rubber plantations of Malaya. On large estates a pest gang is continually maintained, whose sole duty is to prevent and treat disease, for, by the destruction of dead branches and the excision of unhealthy bark, certain diseases can be prevented from spreading beyond the initial stages. On these estates also, eradication of jungle stumps immediately after the rubber trees have been planted is a considerable insurance against heavy losses from root diseases later.

The elimination of alternate host plants plays an important part in the control of some diseases caused by Rust fungi ; for example, eradication of barberry bushes has led to a great reduction of the Black Rust of cereals (*Puccinia graminis*) in some countries. With virus diseases one of the most effective means of control is to destroy the insect carriers of the virus, but, unfortunately, this can only be done in exceptional circumstances.

Plants are usually least liable to disease when they are growing vigorously, but certain mildews and rusts tend to attack most virulently plants which are growing best. By careful selection of soil and situation some diseases can be avoided owing to the resistance of the host under favourable conditions of growth. Suitable manurial treatment may assist in keeping disease at bay: potash in slight excess confers a high measure of resistance against some diseases, and phosphates promote early maturation, which may lead to escape from disease altogether. Efficient drainage of the soil aids in checking diseases which attack the underground parts of plants. By planting at particular times, certain crops can be grown when the germs of disease are not available for infection. Early potatoes are rarely attacked by Blight, not because they are resistant to *Phytophthora infestans*, but because they are grown at a time when blight spores do not exist in the air. Slight changes in methods of cultivation are often sufficient to cause a considerable reduction in attack by disease. Deep

earthing-up of potatoes renders it more difficult for the tubers to be attacked by Blight. Extension of the rotation is a well-known means of reducing the risk of infection by certain soil-borne diseases. The thinning-out of the branches of fruit trees in summer involves less danger of attack by *Stereum purpureum* than when this operation is carried out in winter. Wounds in trees can be protected against parasitic invasion by early treatment with suitable paints or grafting-wax.

The use of fungicides now plays a prominent part in the control of plant diseases. These substances are generally applied in liquid or powder form to the crop before the germs of disease are present in the air, so that many fungicides are preventive rather than curative. Particulars of the fungicides in common use will be found in Chapter XXII. On a small scale, e.g. in greenhouses and nursery beds, toxic substances as well as steam can be applied to kill parasitic organisms in the soil. Many diseases, which, like the Smuts, are seed-borne, can be prevented by treating the seed with hot water or with such fungicides as formalin, copper sulphate, and hydrogen peroxide, although it is preferable to choose seed only from healthy crops, if possible.

In all control measures care must be taken that the treatment suggested is economically feasible, i.e. the increase in value of the crop should be greater than the cost of treatment. At the present time there is so small a margin of profit in cultivating most crops that costly measures for controlling disease are prohibitive.

Legislation on plant diseases

Most civilized countries now maintain an official phytopathological service, and legal enactments have been passed in the endeavour to prevent the introduction of new diseases, to eradicate those recently established, and to check the spread of some of the most serious diseases of long standing. In Britain this legislation, embodied in administrative 'Orders' issued by the Ministry of Agriculture and Fisheries, is based upon the Destructive Insects and Pests Acts, 1877 to 1927. By these means efforts are made to prevent the introduction

of serious foreign diseases into the country, the sale of plants affected by certain diseases is prohibited, and measures are taken to limit as far as possible the spread of such diseases as Wart disease of potatoes, Silver-leaf disease of fruit trees, and Onion Smut. In the United States an extremely rigorous quarantine has been instituted to control the importation of living plants, and many plants may not be imported at all.

CHAPTER II

NON-PARASITIC DISEASES

MANY plant diseases are caused by physiological disturbances which are not due to parasites. Each crop plant has a certain range of soil and weather conditions within which alone it thrives. If the divergence from a proper environment be extremely great, the plant quickly dies; if the environment be only slightly unfavourable, the plant continues to live, but often exhibits some abnormality in appearance, constituting a diseased condition. The life of a plant is so complex that disturbances in function often arise, leading to manifestations of disease.

The symptoms of non-parasitic diseases are generally distinctive, but occasionally they are similar to those caused by parasites. Gummosis in species of Prunus, though often due to attack by micro-organisms, may be caused by other disturbances. Silvering of the foliage of fruit trees in Britain is almost universally due to *Stereum purpureum*, but silvering of the leaves of herbaceous plants is certainly not caused by this fungus and is probably brought about by some non-parasitic derangement. In this case one of the effects of both kinds of disturbance is the same, although the inciting causes are entirely dissimilar.

In this book it is not possible to give a full account of non-parasitic diseases, and only a few of the commoner maladies of this kind will be discussed. The real causes of many of these diseases are still unknown and will probably remain obscure until further progress has been made in our knowledge of the physiology of normal plants.

Unduly low temperatures often give rise to abnormalities which may lead to symptoms of disease and even death. One of the most familiar effects of low temperatures is the sweetening of potato tubers, which is caused by the transformation of starch into sugar at temperatures near freezing-point. Plants .

near the northerly limit of their cultivation frequently suffer
frost injury—the symptoms of which are very diverse—and
may be killed during excessive cold. Cankerous lesions and
cracks in trees are often caused by severe frost. Injury due
to frost is chiefly caused by the withdrawal of water from the
living cells into the intercellular spaces and the formation of
ice crystals there, leading to death of the cells.

Unduly high temperatures also lead to disease and death.
Intense insolation of seedling trees at soil level often causes
collapse of the hypocotyl and subsequent death of the young
plants. This effect has received much attention from foresters
in recent years, as mentioned by Hiley [1].

Flooding or water-logging of the soil, even for short periods,
may seriously affect the growth of crop plants; long-continued
flooding, indeed, leads to the death of most plants that do not
live in swampy regions. Temporary water-logging gravely
interferes with nitrification and may be associated with
yellowing of the foliage, as shown by cereals on poorly drained
soil during a wet spring. Respiration of the roots is always
impeded in a water-logged soil, and if this condition be long
maintained the roots may be completely asphyxiated.

If the atmosphere is extremely humid or if the water-intake
by the roots is greater than the water-loss from the shoots,
local proliferations of the tissues, known as *Intumescences*,
may arise on leaves, young fruits, &c. Leaf intumescences are
usually formed by the elongation and division of the mesophyll
cells. When these lesions are large and widespread, the con-
dition is known as *Oedema* or *Dropsy*.

The commonest effects of drought on plants are well known.
Temporary excessive loss of water generally produces a wilt,
from which the plant recovers on restoration of the water-
balance. Long-continued wilting leads to death. A curious
disease of tomato fruits, known as *Blossom-end Rot*, is due to
temporary water-shortage. In this disease a small water-
soaked spot arises near the stigma end of the fruit, which
enlarges and turns brown. The discoloured region extends
deeply into the fruit and may become invaded by micro-
organisms which induce a soft rot.

Some of the most striking non-parasitic diseases are caused by soil defects. On some soils the foliage is yellowish, a condition known as *Chlorosis*, which may impede growth considerably. In certain parts of England where fruit trees are cultivated on a chalky subsoil, the foliage becomes very chlorotic. This condition is brought about by an excess of lime, which transforms soluble iron compounds into insoluble colloidal substances unavailable to the plant. As iron is necessary for the formation of chlorophyll, deficiency of this element leads to yellowing. On such soils Chlorosis is ameliorated somewhat by covering the soil with herbage, e.g. clover and grass, instead of keeping it in clean cultivation. Soils containing an excess of manganese also cause Chlorosis, again through the iron compounds being rendered difficult of absorption. Chlorosis due to excess of lime or manganese in the soil can sometimes be cured by injecting a solution of iron sulphate into the tissues, or, in some instances, by spraying the foliage with this substance. Another cause of Chlorosis is deficiency of magnesium in the soil, as indicated by Graebner [2]. Magnesium is a necessary constituent of chlorophyll, so that a deficiency of it leads to yellowing. Chlorosis caused in this way is most common on soils leached by heavy rains. As indicated previously, yellowing of the foliage may be associated with defective drainage (often causing deficiency in available nitrogen). The presence of nitrogen in large excess may also induce Chlorosis.

Deficiency of potash in the soil is often accompanied by abnormalities in the foliage. This is very striking in potatoes, the leaves becoming bronzed or yellowish and dying prematurely. *Leaf Scorch* of apple trees has been shown by Wallace [3] and others to be often associated with lack of potash; it can sometimes be prevented by the addition of a potassic manure. With lack of potash water-strain in the leaves is increased, causing scorching, but it is not known exactly how this is brought about. Hatton and Grubb [4] state that the incidence of Leaf Scorch in apples is considerably influenced by the type of stock on which the trees are grafted.

On some alkaline soils oats cannot be grown profitably on account of a disease known as *Grey-leaf*. This trouble has

been investigated in Holland by Hudig[5], and it occurs in some parts of the British Isles. The symptoms are a greyish blotching of the leaves of young plants, which grow poorly and die sometimes at an early stage. This disease can be counteracted by using manures such as superphosphate and sulphate of ammonia, which tend to make the soil acid; it can be prevented more certainly by the addition of about one cwt. of manganese sulphate per acre.

Exanthema of citrus trees is believed to be caused by unsuitable soil conditions, including excessive nitrogen, the presence of a hard pan near the surface, and defective drainage, but its etiology is not clearly understood. According to Floyd[6], who has investigated the disease in Florida, the terminal branches of affected trees are discoloured, gum pockets are formed at the nodes, exudations of gum arise on the older branches, and the fruit is stained. The disease has been reduced in some parts of Florida, and also in Western Australia according to Wickens[7], either by spraying the trees with Bordeaux mixture or by applying copper sulphate in powder form to the soil.

Respiratory troubles often cause serious diseases, as when fruits and tubers are stored in such a way that carbon dioxide accumulates unduly in the surrounding atmosphere. The condition known as *Brown Heart* in apples has been shown by Kidd and West[8] to be caused by asphyxiation of the fleshy part of the fruit when there is a large increase of carbon dioxide and a decrease of oxygen in the surrounding atmosphere, the presence of some oxygen being essential. Apples affected by Brown Heart usually appear sound on the surface, but much of the flesh between the core and the skin is discoloured. During 1921 and 1922 large consignments of apples from Australia were found to be affected by Brown Heart on arrival in England. Kidd and West[8] showed that this arose through the ships' holds being insufficiently ventilated. Respiration of the apples led to a great concentration of carbon dioxide, the danger point being reached when the atmosphere contained about 13 per cent. of this gas. In this connexion it may be mentioned that an increase of concentration of the carbon

dioxide up to about 10 per cent. delays ripening in apples without injury, a fact which may be made use of in a commercial process for the storage of this fruit, as described by Kidd, West and Kidd [9].

The condition known as *Black Heart* of potato tubers is caused by deficiency of oxygen in the surrounding atmosphere, and is often accompanied by abnormal accumulation of carbon dioxide. Bartholomew [10] first associated this disease with overheating, but Stewart and Mix [11] have shown that deficiency of oxygen and increase of carbon dioxide in the atmosphere will produce this condition at as low a temperature as 40° F. Tubers affected by Black Heart may appear normal on the surface, but on being cut open show a black centre.

Another common trouble in stored apples is *Superficial Scald*, characterized by discoloration of the skin, and, in severe cases, by a browning of the flesh below the skin. C. Brooks, Cooley, and Fisher [12] have shown that this is caused by the accumulation of volatile esters, formed in ripening, in the surface layers of the fruit. Superficial Scald often develops after removal of the apples from storage. Low temperatures tend to check the development of this trouble, and the authors [13] mentioned have demonstrated that it can be prevented by keeping the apples wrapped with paper soaked in mineral oil during storage. The oil absorbs the volatile substances, which lead, on accumulation, to the discoloration. There are other non-parasitic diseases of stored apples which are sometimes called Scald, but these have not been fully investigated yet.

Internal Breakdown is another affection of apples in middle or late storage life, characterized by a soft, brownish disintegration of the flesh, and later by a darkening of the surface; it is sometimes difficult to distinguish Internal Breakdown from Brown Heart, but in the latter the discoloured flesh is firm and sharply delimited from the healthy tissues. Internal Breakdown is more prevalent in cold storage than in ordinary storage, and, according to Kidd and West [8], it is not associated with high carbon dioxide or low oxygen concentrations in the surrounding atmosphere.

Bitter-pit is a commonly occurring functional disease of

apples both on the tree and in storage, the cause of which is at present unknown. In England it is most prevalent in hot, dry summers. The flesh of an apple affected by Bitter-pit contains small, brown spots, which are most abundant just under the skin. The spots near the surface appear as depressions or pits, brown or brownish-green in colour, on the outside of the fruit. The brown spots are dry and consist of collapsed cells which contain starch grains. Different kinds of apples vary considerably in susceptibility to Bitter-pit, and the disease differs in intensity from season to season. Apples apparently free from Bitter-pit at the time of gathering often develop it in storage. The extensive literature on this disease has been critically summarized by Smith[14]. He points out that heavy irrigation towards the end of the season and severe pruning predispose to Bitter-pit, and that fruit gathered in an immature condition is especially prone to develop this defect. He suggests that for the export of apples from Australia late picking combined with rapid cooling may be expected to reduce considerably the development of Bitter-pit during shipment.

Glassiness or *Water-core* of apples may develop as the fruits reach maturity on the tree. In this condition parts of the flesh appear to be glassy, and in extreme cases portions of the surface may be semi-translucent. Glassiness is due to the exudation of water from the cells of the flesh into the intercellular spaces, but the inciting cause is unknown. This condition often disappears during storage.

Gaseous fumes, particularly coal smoke and gas used for lighting, are very injurious to plants. The effect of coal smoke is to cause browning of the whole or part of the leaf lamina, premature shedding of the leaves, and sometimes death of the whole plant. Conifers and lichens are particularly sensitive to smoke injury, and in the industrial districts of the Midlands these plants do not occur or cannot be grown on account of the smoky atmosphere. Dicotyledonous trees are less subject to smoke injury, but the Ash is very sensitive. The most toxic constituent of coal smoke is sulphur dioxide, which destroys chlorophyll. Coal gas, on burning, forms small quantities of sulphur dioxide and other toxic gases which

seriously injure plants in rooms lit with this illuminant. The effect of smoke-pollution on the vegetation around Leeds has been studied by Crowther and Rushton [15], and a full survey of the problems of smoke injury to plants has been made by Stoklasa [16].

The constituent of coal gas most deleterious to plants is ethylene, according to Crocker and Knight [17] and Harvey and Rose [18]. Minute traces of ethylene have an injurious effect upon many plants, causing leaf-fall, preventing the buds from opening, and ultimately causing death in some instances. Small amounts of ethylene in the surrounding atmosphere promote ripening of certain fruits in storage. If coal gas leaks out into the soil it may seriously damage the roots of trees, partly by asphyxiation and partly by the influence of its toxic constituents. A row of elm trees in Cambridge was killed recently owing to an escape of gas from a main alongside.

Excessive wounding of certain kinds of trees may result in curious diseases. In the extraction of latex in the rubber plantations of the eastern tropics the bark of the trees is cut away in thin strips almost daily. In the early development of this industry the trees were tapped too vigorously, one of the effects of which was the development of the disease known as *Brown Bast*. This affection is marked by a brown discoloration of the phloem and cortex on the exposed tapping surface, coupled with the cessation of the flow of latex from the diseased tissues; at a later stage numerous woody burr-knots develop in the renewing bark, which may render it untappable subsequently. Most plant pathologists now consider Brown Bast to be a functional disease. Sharples [19], who has studied it in Malaya, is of the opinion that Brown Bast is brought about by the withdrawal of excessive quantities of water from the tree during heavy tapping. If the disease is observed in the early stages and tapping is stopped, it does not progress downwards, and after a period of rest the trees can be tapped again. In severe attacks bark affected by Brown Bast is often excised. With the more conservative systems of tapping now customary this disease is not likely to be so prevalent in the future as in the past.

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CHAPTER III

VIRUS DISEASES

ALTHOUGH undoubtedly existing for ages past, virus diseases of plants have been clearly recognized and studied only in recent years, but some of them now rank among the most destructive plant diseases. Apart from a few records, no parasite can be seen in the diseased tissues, but, in view of the infectious nature of these diseases and of the ability of the infective 'principle' to multiply within the host, they are often supposed to be caused by organisms which are ultra-microscopic in size, at any rate in some phases of their life-history. In several of these diseases the infective 'principle' will pass through Berkefeld and Chamberland filters without loss of virulence. Kunkel[1] and Cook[2] have discovered curious bodies in the cells of maize and sugar-cane affected by mosaic disease, which they consider may possibly be living organisms, but Kenneth Smith[3] is of the opinion that somewhat similar bodies in potato mosaic are degeneration products of the host protoplasm. Rawlins and Johnson[4] also describe inclusions of various kinds in the cells of the chlorotic areas in tobacco mosaic. These inclusions have been studied by Goldstein[5], who considers that the 'x' bodies in mosaic tissue represent some phase in the life-history of a parasite; Hoggan[5a], however, after further investigation of these 'x' bodies, inclines to the view that they are not of the nature of a causal organism. Link, Jones, and Taliaferro[6] have shown that a myxomycete, *Plasmodiophora tabaci*, associated with tobacco mosaic and sometimes also with healthy leaves, is certainly not the cause of this mosaic.

Olitsky[7] is the only person who claims to have obtained multiplication of a virus *in vitro* (tomato mosaic). Henderson Smith[8] and others have repeated Olitsky's experiments, but without success.

Against the belief in the parasitic nature of virus diseases may be cited Johnson's[9] result that a form of mosaic in tobacco may be induced by injecting juice of supposedly healthy potatoes. It may be, however, that the potatoes, while showing no symptoms, were 'carriers' of the disease.

In many virus diseases the infective 'principle' can be withdrawn with the plant juice and kept for a long time (fifteen months at least in tobacco mosaic) without losing virulence. The virus, so extracted, can be diluted many times without reduction in infectivity. Dried leaves of tobacco affected with mosaic

retain their virulence for at least three years. Allard[10] states that the virus of tobacco mosaic is destroyed quickly by 80 per cent. alcohol, although it is not killed by 50 per cent. alcohol; it is destroyed speedily by 4 per cent. formalin. This virus is quickly killed at temperatures near 100° C., but it sometimes withstands heating to over 80° C. for five minutes.

Although the tobacco mosaic disease can be transmitted by touching an abrasion in a healthy plant with sap containing the virus, and other mosaic diseases can be transmitted by injection, many virus diseases can only be transmitted by budding or grafting, or by insect agency. Baur[11] has shown that certain types of infectious chlorosis can be transmitted by grafting, but not by injection of affected sap. Under natural conditions many virus diseases are now known to be carried from plant to plant by insects, of which aphides, jassids, and plant bugs are the most important. Sometimes a period of incubation for the virus in the insect appears to be necessary before it can transmit the infection to a healthy plant. For instance, Kunkel[12] states that the adult jassid, *Cicadula sexnotata*, which is the vector of ' Aster Yellows ', cannot transmit the disease until six days after feeding on an affected plant. These diseases are only rarely transmitted by seed. Some viruses have been shown to travel in the plant chiefly by way of the phloem and parenchyma.

The symptoms induced by virus diseases are very diverse, but they generally result in a systemic infection of the shoot system, which causes the new growth to be more or less transformed; shoots fully developed at the time of infection are only rarely modified. The roots are often permeated by these viruses. Only occasionally does a virus disease kill the plant outright, as e. g. in ' Spinach Blight ' (mosaic), as described by McClintock and Smith.[13]

Many virus diseases of crop plants have now been described, but further research may show that these are not all distinct, particularly in the case of the mosaic diseases. Several distinct viruses may, however, affect the same host, and at one and the same time.

The symptoms of any specific virus disease may vary somewhat in different varieties of the host, and a variety, although infected, may show no obvious sign of disease. Such a variety, or, it may be, other species, acts as a ' carrier ' of the disease. Some ' carriers ', however, show symptoms of disease when the environment is changed. Certain environmental conditions may also lead to the masking of disease symptoms: thus Rawlins and Johnson[4] state that below 18° C. and above 34° C. the symptoms of tobacco mosaic do not appear.

Peach Yellows

This serious disease of peach trees in certain parts of North America was first investigated by E. F. Smith[14], who found

that the disease could be communicated by inserting a bud from an affected tree into a healthy one, but not by the injection of juice from a diseased tree. The natural means of dissemination are still unknown, notwithstanding the fact that the disease sometimes spreads epidemically.

Affected leaves are usually pale green or yellow, narrower than normal, more or less drooping in habit, and rolled or curled at the margin. Other characteristic features of the disease are the production of slender, branched, erect shoots, and premature ripening of the fruits, which are often speckled with red. A tree once affected inevitably dies in time. The virus is present in the apparently healthy parts of trees only visibly attacked in a single branch. Cook [15] states that translocation of starch is more or less inhibited from the affected leaves. The only means of checking the spread of Peach Yellows is to destroy affected trees as soon as seen.

'Little Peach' is a somewhat similar destructive virus disease in North America, the distinguishing symptoms being small fruits, which ripen late, coupled with absence of the slender twigs present in Peach Yellows.

These diseases of the peach have not yet been recorded in England.

Curly Top of Sugar-beet

So far as is known at present this disease occurs only in America, especially in the western parts of the United States. In affected plants the leaves are markedly curled and rolled, the veins being thickened and distorted on the under surface, and there are many fine, fibrous roots. The disease is most serious when the seedling is attacked. Plants infected at a late stage exhibit the symptoms only in the new growth. Badly affected plants may die prematurely or remain stunted. Garden beet and mangolds are sometimes attacked as well as sugar-beet.

The disease is disseminated apparently solely by the leaf-hopper, *Eutettix tenella*, Baker, which, once infected, generally remains a vector throughout life. A leaf-hopper does not become infective until 4 to 24 hours after feeding on a

diseased plant.   The symptoms appear in about 7 to 14 days after the plant has been fed upon by the vector.   Certain annual weeds can also become affected, and these may be a source of infection in spring, as the insect feeds upon them too.   Direct inoculation with the juice of an infected plant is sometimes successful.

According to Carsner and Stahl [16] there is marked necrosis of the phloem throughout the affected plant, and in this respect the disease is similar to ' Leaf-roll ' of potatoes.

FIG. 1.   Potato ' Leaf-roll ' (var. ' Edzell Blue '). (*Kenneth Smith.*)

Sugar-beet mosaic, which is widespread in Europe, is a distinct disease of less serious character than Curly Top.

Leaf-roll of Potatoes

Although prevalent for many years this disease has only comparatively recently been recognized as being infectious. In some parts of England it is one of the most serious virus diseases that affect potatoes, and it is largely responsible for the ' degeneration ' of this crop described by earlier writers.

The symptoms are a rolling upwards and thickening of the leaflets, which become brittle, a dwarfing of the plant, and

the production of few tubers, small in size. When a plant is first infected, only the upper leaves become rolled, as the virus, although soon permeating the entire plant, does not usually modify organs formed before infection. If tubers from such a plant are grown again in the following year, the lower as well as the upper leaves will be rolled and otherwise modified. If seed tubers are again saved from such plants there is progressive degeneration, which sometimes results in a nearly worthless crop. The seed tubers of plants showing secondary symptoms are often still hard when the crop is lifted. In some varieties, e. g. ' President ', the disease is accompanied by a pinkish discoloration of the leaflets.

The rolling and brittleness of the leaves is chiefly due to an abnormal accumulation of starch, which is not translocated away as in healthy leaves. Quanjer [17] and Artschwager [18] have found that the phloem of the shoots of affected plants is disorganized, and for this reason the disease is sometimes called ' phloem necrosis '. Schultz and Folsom [19] state that in the United States the disease is sometimes accompanied by necrotic areas in the tubers, but this has not been observed in England.

Infection is spread from plant to plant by insects, of which aphides are the most important, but, according to Murphy [20], capsid bugs (*Calocoris bipunctatus*) and jassids (*Typhlocybe Ulmi*) also transmit the virus. Murphy states that the disease is occasionally carried over in the true seed. There is as yet no proof that infection takes place from the soil. Although the disease spreads chiefly under field and garden conditions, Murphy and McKay [21] have shown that it may be disseminated by aphides during sprouting of the seed tubers before planting.

Leaf-roll is less prevalent in certain parts of Scotland (particularly the north) and in Ireland than in most parts of England, and long before the nature of the disease was understood it was customary in England to obtain seed potatoes each year, or every alternate year, from more favourable areas. The infrequency of Leaf-roll in Scotland and Ireland may, perhaps, be correlated with lack of aphides and other insects there on account of a cooler and wetter climate.

The only way to ensure freedom from this and other virus diseases of the potato is to take care that the seed tubers are obtained from healthy stocks. At present the best growers in Scotland destroy plants visibly affected by virus diseases and lift the healthy plants early. If, however, more than a small percentage of plants has been affected by Leaf-roll, no part of the crop should be saved for seed. The time may come when selected stocks of potatoes will be grown in isolation, i. e. remote from other potatoes, for seed purposes. Aphides on sprouting potatoes may be destroyed by fumigation, e. g. with tetrachlorethane.

The following varieties of potatoes grown in Britain are very susceptible to Leaf-roll: 'Midlothian Early', 'May Queen', 'British Queen', 'Up-to-date', 'King Edward', and 'President'. The following are resistant: 'Epicure', 'Resistant Snowdrop', 'Arran Chief', and 'Great Scot'. Some of the so-called resistant varieties are probably merely tolerant to the virus, and may perhaps act as 'carriers' of the disease. There are grounds for thinking that the symptoms of Leaf-roll may be suppressed under certain conditions.

Mosaic Diseases of Potatoes

There are several diseases of potatoes of the mosaic class which can all be distinguished readily from Leaf-roll, but which can be distinguished from one another only with difficulty. So far, the investigators of these diseases have not adopted a uniform nomenclature, and this sometimes leads to confusion in diagnosis. Furthermore, the symptoms of these diseases vary considerably in different varieties. All of these diseases are infectious and are chiefly spread by aphides. They are all systemic, and several lead to marked degeneration. The mode of control of these mosaic diseases is essentially the same as that described for Leaf-roll of potatoes. Some varieties which do not show marked symptoms under ordinary conditions are known to be 'carriers' of the viruses, and under some conditions the symptoms may be suppressed even in varieties which usually show them.

Common Mosaic

The symptoms, which may be suppressed in hot, dry weather, are the presence of yellowish, mottled areas in leaves. There is no necrosis of the phloem. In many varieties this form of disease does not lead to serious reduction in yield. Some observers, however, think that Common Mosaic leads in some varieties to 'Curly Dwarf', a form of disease characterized by intense dwarfing of the plant, wrinkling of the leaves, and a worthless crop.
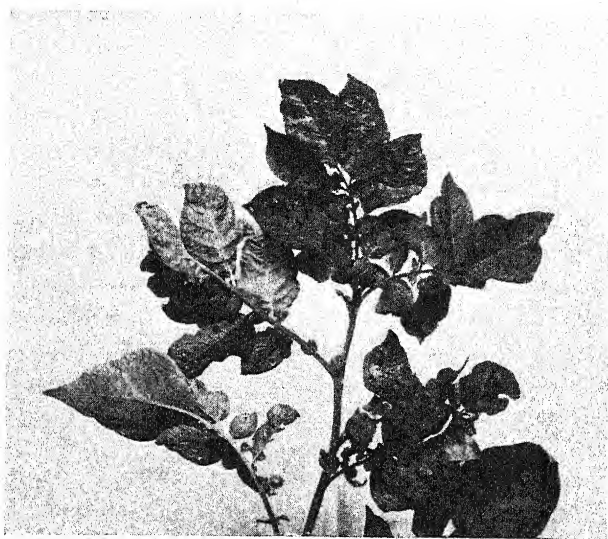

Fig. 2. Potato 'Crinkle' (var. 'Myatt's Ashleaf').

Crinkle

As described by Quanjer[22] the leaflets show lighter areas and are markedly corrugated, with the margins and tips curled downwards. The lower leaves have fine, dark stipples, and they turn yellow and drop prematurely. Murphy[23] considers that Crinkle may cause marked deterioration.

Stipple-streak

According to Atanasoff[24], this is one of the most serious virus diseases, leading to rapid degeneration. In the primary form of the disease the upper leaves show brown, angular spots, which include parts of the veins, and subsequently these leaves wilt and die prematurely; the stems also show dark stripes. The

secondary symptoms are dwarfing and premature death of the plants, the leaves of which are brittle and marked with brown spots. Such plants produce few, very small tubers, which are rough, split, and often discoloured. There are necrotic areas in the pith and cortex of the stems, and some of the eyes of the tubers may be destroyed.

Leaf-drop Streak or Leaf-drop

The most marked feature of this disease is the premature fall of the lower leaves. There are dark streaks on the stems and petioles, but not on the laminae. Perhaps more than one disease is included in this category.

Tomato Mosaic

A number of mosaic viruses may affect the tomato plant (e. g. tobacco and cucurbit mosaic), and it is not yet certain whether there is a specific 'tomato mosaic'. The disease generally known as Tomato Mosaic is prevalent wherever tomatoes are grown on a large scale. Affected leaves are often mottled, light-green or yellowish areas being intermingled with normal green ones; owing to the slower growth of the light-coloured areas the surface is very irregular. According to Bewley [25] other leaf symptoms are a deep yellow spotting of the lamina, distortion of the margin, blistering of the surface, and a reduction of the lamina to little more than the midrib. Brown streaks may occur on the stems and petioles. In the United States the fruit of diseased plants is frequently badly blotched and unsaleable, but this defect is uncommon in England. Plants which are affected when young may be considerably stunted. Under greenhouse conditions the effect of the disease is most serious after the growth of the main stem is stopped, when, owing to weakness of the lateral shoots, the crop may be negligible.

The virus spreads to every part of the plant except perhaps the seed, even though only a few leaves may be visibly affected. The virus affects Petunias and wild species of Solanum; it can be communicated to potatoes by grafting, but is probably not identical with either of the virus diseases of that crop.

Tomato mosaic is conveyed by aphides and other sucking

insects, and may perhaps be transmitted by careless handling of healthy plants after contact with diseased ones.

Bewley [25] states that individual plants of susceptible hosts may act as 'carriers' of the virus.

Diseased plants and weeds likely to harbour the virus should be destroyed, and insect pests should be controlled, especially in greenhouses.

According to Vanterpool [26] and Dickson [27] the Stripe or Streak disease of tomatoes, which is sometimes considered to be due to *Bacillus Lathyri*, is caused by a mixed infection of tomato or tobacco mosaic and potato mosaic.

Tobacco Mosaic

This mosaic causes heavy losses in tobacco cultivation. It was one of the first virus diseases of plants to be studied, and it has been more fully investigated by Beijerinck [28], Iwanowski [29], Allard [30], Chapman [31], McKinney [32], and others than other mosaic diseases. The virus is closely related to, and may be identical with, that of tomato mosaic; it can be communicated to the tomato and other plants of the Solanaceae. The virus is transmitted by insects, including the aphis *Myzus persicae*, and if only a slight abrasion in a healthy plant is touched with juice from an infected plant, mosaic is thereby induced. The effect of this virus on the tobacco plant has been carefully studied by Goldstein [5], who points out that the symptoms vary according to the stage of development of the leaf at the time of infection.

Several different viruses, which have been distinguished by Johnson [24a], may produce mosaic symptoms in tobacco.

Cucumber Mosaic

This disease affects many plants of the Cucurbitaceae, but is most serious on the cucumber. The stem internodes are short, the leaves are mottled and wrinkled, the flowers are small and pale, and the fruits mottled and sometimes disfigured by dark green, wart-like outgrowths. The disease may be carried over in the seed. In England, Bewley [25] has noticed that some

cucumber plants, not otherwise seriously affected, may have bright yellow patches in the leaves ('Aucuba' type of mosaic), but this may be due to a different virus.

According to Doolittle [33] the melon aphis and certain beetles are the chief vectors, and he considers that in the United States allied wild plants assist in the overwintering of the virus. He found that filtration of the juice of mosaic plants through a Berkefeld filter did not eliminate infectivity, although Chamberland filters rendered the filtrate innocuous. He states also that juice from a diseased plant loses its infectious character after a week, and that it will not withstand drying, heating above 70° C., or treatment with antiseptics except 5 per cent. chloroform and toluene.

In England the variety 'Butcher's Disease Resister' is only slightly affected by this disease.

Raspberry Mosaic

The varieties 'Superlative', 'Baumforth B', and 'Bath's Perfection' are very susceptible to this virus in Britain. The leaves of affected plants are mottled with yellow and curl downwards, and the canes are very dwarf and bear little fruit. Harris [34] states that the disease may have a very serious effect on the cropping of some varieties. The virus extends throughout the plant, so that canes propagated from diseased stools are also affected. In plantations the disease spreads from plant to plant, although it is not yet known how this is effected. The disease can be artificially transmitted by grafting. Badly affected stools should be destroyed and only healthy ones should be used for propagation.

In the United States, Bennett [34a] has distinguished several different mosaic diseases in raspberries, and further investigation may show that more than one type of raspberry mosaic occurs in Britain.

Hop Mosaic

Salmon and Ware [35] have pointed out that this disease is a considerable menace to hop-growing in England. Affected

leaves are brittle, mottled with yellow, and more or less curled, with recurved margins; the bines have shortened internodes and a limited growth, usually of four to six feet. Dwarf bines do not produce 'hops', and when the affected bines are taller the hops are malformed. Diseased bines may die during the summer, and affected 'hills' may die in the course of a year or two. The disease is very infectious; it is presumed that aphides play an important part in dissemination. Some varieties, e.g. 'Fuggles', are 'immune', but these may act as 'carriers' of the virus, as shown by Thrupp [36]. Hop mosaic can be transmitted by grafting. Affected 'hills' should be destroyed in the early summer, and, as far as possible, only plants in completely healthy gardens or nurseries should be used for propagation.

'Nettlehead' is another virus disease of the hop plant, which causes stunting of growth and sterility, but it does not kill the plant outright and does not spread so rapidly as the mosaic disease.

Sugarcane Mosaic

First discovered in Java, this disease has now spread to almost all sugarcane-growing countries. In many commercial varieties it causes a marked reduction in yield. The disease is characterized by a mottling of the young leaves, the yellowish patches sometimes giving a striped effect; as the leaves age, the mottling tends to disappear; discoloured stripes sometimes occur on the stems, which are often thinner and shorter than usual. According to Cook [37], the choroplasts in the yellowish areas are undeveloped rather than disintegrated. The virus passes down the stems, and it may perhaps extend into the rhizomes.

The disease is primarily spread by propagating from the tops of affected canes, and Brandes [38] has shown that the virus is transmitted by means of *Aphis maidis*. According to Brandes and Klaaphack [39] the same virus causes mosaic in maize, sorghum, and several wild grasses.

Sugarcane varieties show great differences in susceptibility, there being every gradation between extreme susceptibility

and 'immunity'. No thick varieties of cane have yet been found to be 'immune', but the thin variety 'Uba' has remained free from mosaic both in Natal and in Jamaica, according to Storey [40] and Hansford and Murray [41].

Control is to be sought by the use of 'immune' and resistant varieties, coupled with care in propagating susceptible varieties only from healthy plants.

Streak Disease of Sugarcane and Maize

Storey [42] has shown that the Uba cane, although 'immune' from mosaic, is attacked in Natal by another virus disease, which is distinguished under the name of 'Streak'. The same virus affects other varieties of cane (some of which are susceptible to mosaic), maize, and certain grasses.

Streak disease is characterized by the presence of narrow, colourless areas along the leaf veins, most readily recognized in the young leaves, whereas the markings of mosaic are diffuse and spread over several veins. Storey has found that the vector is the jassid, *Balclutha mbila*. The disease is also spread in sugarcane by propagating from diseased tops, but it is not seed-borne, at any rate in maize. Considerable reductions in yield are caused by this disease in the Uba cane.

According to Storey, a jassid becomes infected by feeding on diseased tissues for fifteen minutes, but there is an incubation period in the insect before it can transmit the virus. Certain individual jassids do not transmit the virus although previously fed on diseased tissues. If the tips of young leaves be inoculated with the virus, the symptoms of the disease appear at the base of the leaf within forty-eight hours. Storey [42a] states that the virus moves through 40 cm. of leaf in two hours at 30° C., and that its movement is not retarded by severing the main veins.

'Reversion' of Black Currants

Evidence is accumulating that this disease is caused by a virus, for it is infectious and it can be transmitted by grafting; in nature it is transmitted by the mite which causes 'Big Bud'

of black currants, as shown by Amos, Hatton, Knight, and Massee [43].

The symptoms of ' Reversion ' concern chiefly the leaves and flowers. Lees [44] has pointed out that the leaves of reverted shoots have fewer sub-main veins than usual (i. e. four or less), with the margin less serrate than in healthy leaves; the lamina is sometimes considerably elongated relatively to its breadth. Reverted shoots give rise to abnormal flowers which are devoid of the hairy ' bloom ' characteristic of healthy ones, and which rarely produce fruit. The disease may first appear on a single branch, but it spreads throughout the bush and is present in apparently healthy branches. The first-formed leaves of a reverted branch are normal, but those formed somewhat later show the characteristic symptoms; leaves produced towards the end of the season may also appear normal. Entire plantations in England have had to be destroyed in recent years in consequence of this disease.

The utmost care should be taken in propagation to take cuttings only from indubitably healthy bushes which have been inspected in June. If the disease appears in beds of cuttings or in young plantations the affected plants should be destroyed. ' Big Bud ' mite can be controlled by spraying the bushes with lime-sulphur when the buds open.

Healthy currant shoots sometimes show 'reverted' characters in the lowest leaves. This is not true Reversion, but 'false reversion ', and is caused by injury, especially to the terminal bud.

Bunchy Top of Bananas

As described by Darnell-Smith [45] the first symptom of this disease is the development of dark green streaks on the under side of the midrib of a young leaf, and the ultimate effect is a dwarfing of the plant and the bunching together of the upper leaves, which become rigid and brittle. Affected plants are worthless. 'Bunchy Top' occurs in bananas in Australia [46] and Fiji, and in Manila hemp in the Philippines.

No visible parasite is associated with the disease, and, as it has been shown to be transmitted by the aphis *Pentalonia nigronervosa* by Goddard [47] in Australia, and by Ocfemia [48] in the Philippines, 'Bunchy Top' is considered to be a malady of the virus type. Symptoms appear about a month after infected aphids have fed

upon a plant. Inoculations with sap from diseased plants have not hitherto reproduced the disease.

Government action is being taken in Australia to try to prevent the introduction of the disease into non-infected areas.

Rosette Disease of Peanuts.

This disease of peanuts or groundnuts (*Arachis hypogaea*) causes serious losses in South Africa, and what may prove to be the same disease is also prevalent in tropical Africa, Java, and India. The disease is characterized by a yellowing of the leaves and extreme stunting, and when infection occurs early in a plant's growth no seeds are produced. The disease has been investigated in South Africa by Storey and Bottomley (49), who consider it to be caused by a virus which is transmitted by *Aphis leguminosae*, Theo. In South Africa the virus is believed to be carried over in diseased plants which survive the winter. Peanut Rosette may spread epidemically during early summer. Storey and Bottomley state that, in an average season, the crop largely escapes infection if planted as early as possible ; they suggest the destruction of surviving plants during the winter.

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CHAPTER IV

DISEASES CAUSED BY BACTERIA

BACTERIA are extremely minute, unicellular organisms, devoid of chlorophyll, which reproduce very rapidly by fission. The cells may be rod-like (Bacterium, Bacillus, Pseudomonas), spherical (Coccus) or curved (Vibrio, Spirillum), and they may be joined together temporarily in filaments or other aggregates. Most bacterial cells are devoid of a typical nucleus. Many bacteria are motile during part of their life, the organs of locomotion being slender protoplasmic threads called flagella. Endospores, usually one in each cell, are formed in some species, but none of the bacteria pathogenic to plants, except possibly *Pseudomonas seminum*, Cayley, produce spores as far as is known.

Practically all bacteria pathogenic to animals and plants can be cultivated saprophytically, and it is by their cultural behaviour alone that these organisms can be adequately characterized.

Bacteria pathogenic to plants have been studied intensively only in comparatively recent years. These organisms invade plants chiefly through wounds or through the stomata. They live principally either in the vascular tissues, causing wilts, or in the parenchyma, which they may rot or cause to become hypertrophied.

There is considerable confusion at present as regards generic limitations in the nomenclature of this group of organisms. In Migula's classification non-motile, rod-like forms are referred to the genus Bacterium ; motile, rod-like forms with flagella at one end to Pseudomonas ; and motile rod-like forms with flagella all round the cell to Bacillus.

In E. F. Smith's scheme, the Bacterium of Migula becomes Aplanobacter, and Pseudomonas becomes Bacterium.

The Society of American Bacteriologists propose, in Plant Pathology, the name Phytomonas for both the Bacterium and Pseudomonas of Migula, and Erwinia for Migula's Bacillus, but this classification has not yet been generally adopted.

Lastly, in other branches of Bacteriology the system of Lehmann and Neumann [1] is being generally adopted, and it would appear desirable that the same system should be used for bacteria pathogenic to plants. In this system, rod-like forms devoid of endospores, whether motile or not, are placed in the genus Bacterium. On this basis there would be a single generic name, Bacterium, for all bacteria pathogenic to plants so far described, with the possible exception of *Pseudomonas seminum*, Cayley. In view of this confusion in nomenclature the name of the disease rather than that

of the pathogen is given first in the following pages, the scientific name appended being that which is most commonly used.

The standard books on Bacterial Diseases of Plants are those by the chief pioneer in this branch of Plant Pathology, Erwin F. Smith (*Bacteria in Relation to Plant Diseases*, Carnegie Institution of Washington), and *An Introduction to Bacterial Diseases of Plants*, W. B. Saunders, Philadelphia.

In the following pages complete diagnoses of the bacteria are not given ; for these the reader should consult the above books or the original papers.

Fire Blight

(*Bacillus amylovorus*, (Burr.) Trev. = *Erwinia amylovora*, Soc. Amer. Bact.)

Bacillus amylovorus is rod-like, averaging $1.5 \times 0.6 \mu$; cells generally single, but occasionally in short chains, with 2–4 peritrichous flagella ; it is non-odorous, non-acid-fast, Gram-negative, non-nitrate reducing, more or less viscid, slowly liquefying gelatine ; surface colonies on agar plates white, more or less opalescent, with an entire margin ; submerged colonies smaller with a hazy margin.

Fire Blight is a destructive and widespread disease of pear and apple trees in North America. Waters[2] has reported its occurrence in New Zealand ; it occurs also in Japan. It is unknown in Britain, and the records of it in Switzerland and Italy are of doubtful authenticity. It occasionally affects stone fruit trees, and attacks the English hawthorn in New Zealand and certain other wild hosts in North America.

The bacterium affects many parts of the tree, causing a blight of the blossoms, leaves, fruit spurs, and twigs, a spotting and rot of the fruit, and cankers and a collar rot in the stems. The leaves on affected spurs turn brown and remain attached, giving the appearance as if scorched by fire. The organism may invade uninjured tissues, or, more often, may enter through wounds, frequently those caused by insects. It is chiefly intercellular, but may invade the parenchyma cells themselves and the vascular tissues. It causes disintegration of the host cells, and, in pear fruits, a soft rot. If cankers spread seriously in the main stem, the whole tree may be killed. In spring and summer the bacteria ooze out of the diseased tissues in slimy masses.

As far as is known at present, the organism lives over the

winter only in the stem cankers. Waite [3] has shown that the bacteria which ooze out from these in the spring are carried by insects (flies, bees and wasps) to the flowers, where infection occurs through the nectaries. Subsequently, the disease is transmitted by these and other insects to the twigs and other parts. The disease is most prevalent in warm, humid seasons, especially when growth has been somewhat premature.

The disease can be reduced greatly by the excision of dead twigs and cankers. Day [4] recommends that the surgical instruments and the cut surfaces should be sterilized with the following disinfectant:

Mercuric chloride	.	.	.	.	1 oz.
Mercuric cyanide	.	.	.	.	1 oz.
Glycerine	.	.	.	.	3 galls.
Water	.	.	.	.	1 gall.

Reimer [5] states that *Pyrus ussuriensis* and *P. calleryana* are very resistant to the disease, and recommends that they be used as stocks for pears instead of those employed hitherto. In New Zealand hawthorn bushes near pear and apple plantations should be destroyed if possible.

Crown Gall

(*Bacterium tumefaciens*, Smith and Townsend)

Bacterium tumefaciens is rod-like, $1.2–5 \times 0.6–1 \mu$ in plant tissues, $2.5–3 \times 0.7–0.8 \mu$ on agar cultures; cells single or in pairs, with 1–3 polar flagella; it is aerobic, non-acid-fast, Gram-negative, non-nitrate reducing, not liquefying gelatine; surface colonies on agar plates small, circular, somewhat raised, wet-shining, translucent.

Crown Gall is a widespread disease of a great range of cultivated plants, especially fruit trees, and is characterized by the presence of large galls on the stems and roots, particularly near soil level. The galls may remain soft or become woody. They occasionally produce roots, or may rarely proliferate into leafy shoots. Another form of the disease is seen in the development of large numbers of small, wiry roots ('Hairy Root'), but this type of affection is very rare in Britain.

Cavara [5a] first showed that Crown Gall was caused by a bacterium. This was amply confirmed by E. F. Smith and

Townsend [6], who named the organism *B. tumefaciens*.   Much of our knowledge of the disease is due to Smith and his colleagues [7].   The abnormal development of the host tissue is caused by stimulation induced by the presence of this organism. According to Robinson and Walkden [8], most of the bacteria are present on the outside of the gall, and those within the tissues are chiefly in the intercellular spaces.   Secondary galls may
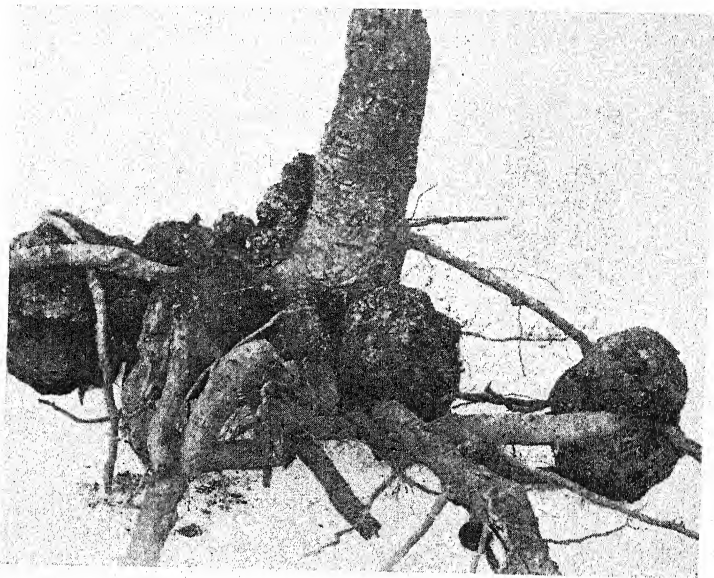
Fig. 3. ' Crown Gall ' of apple.   ¼ natural size.   (*W. J. Dowson.*)

arise at some distance away from a primary centre of infection, chiefly by movement of the bacteria along the intercellular spaces or other tracts.   In general, the organism isolated from one kind of plant will infect most other kinds of plants susceptible to Crown Gall.

B. tumefaciens occurs in the soil and causes infection most commonly through wounds.   The presence of Crown Gall in certain fruit trees, e.g. apple, may be partly correlated with the method adopted for propagating the stocks vegetatively. In removing these from the stool-beds, wounds are made which are very liable to infection.   In the United States, where the

junction of stock and scion is, usually buried below soil level, galls often arise at the place of union. Wormald and Grubb [9] state that the various kinds of apple stocks exhibit marked differences in susceptibility to Crown Gall, Type VII and No. 50 of the East Malling Research Station being extremely susceptible. The same authors point out that the nature of the scion influences markedly the relative susceptibility of the stock. In roses, Manetti stocks are also very susceptible to Crown Gall.

There is considerable difference of opinion as to whether or not the presence of Crown Gall severely affects the growth of fruit trees. The galls often appear to exercise no adverse effect, but at other times they probably cause some injury. Occasionally in other plants, e.g. sugar-beet, Crown Gall may be very harmful and may even cause death when inoculated into the growing-point.

Riker and Keitt [10] have shown that galls, simulating Crown Gall, may arise at moist wounded surfaces, which are proliferations not induced by bacterial invasion. Such overgrowths have often been mistaken for Crown Gall.

Control of the disease under nursery conditions is to be sought in the use of clean land and resistant stocks, and by the treatment of tissues exposed to infection with some protective substance, e.g. soft grafting wax. Under no circumstances should stocks affected by Crown Gall be planted in nurseries.

Citrus Canker

(Pseudomonas Citri, Hasse)

This disease was introduced from the Orient into Texas and Florida about 1911, and it has also occurred recently in S. Africa and Australia. It is most serious on the grape fruit and the lime, but is less harmful to the sweet orange. The causal organism was first described by Hasse [11].

The disease affects the leaves, twigs, branches, and fruit. On the leaves the disease appears as small yellowish spots, first visible on the under surface, which become brown, raised, more or less corky, and surrounded by a yellow halo. The symptoms on the fruit are similar, and cankers are formed in the twigs and branches.

The bacterium causes infection by way of the stomata, when these are covered by a film of water. Fawcett and Lee [12] state that the disease is only active when the mean temperature is above 68° F. and rain is well distributed.

By careful quarantine measures it should be possible to prevent the introduction of the disease into new countries. If the disease is merely slight, affected trees should be destroyed, but where it is widespread only the more resistant kinds of citrus fruits should be grown.

Bacterial Wilt of Cucumbers

(*Bacillus tracheiphilus*, E. F. Smith)

Bacterial Wilt is a rather uncommon disease of cucumbers grown under glass in England. It is much more frequent in the United States, where it affects also the pumpkin, squash, and muskmelon. The disease and the causative organism have been fully described by E. F. Smith [13]. The first sign of disease is the wilting of one or more leaves, which may be accompanied by yellowing; the wilt extends, and usually the whole plant quickly dies. Upon cutting across a diseased stem a sticky, white, bacterial slime exudes from the vascular bundles, the bacteria being found chiefly in the vessels. Weakly plants are particularly liable to attack. In the United States the disease is transmitted by cucumber beetles, and Bewley [14] suggests that the cucumber woodlouse probably disseminates it in England. The disease is most serious at low temperatures, an average temperature of 90° F. checking it greatly.

Angular Leaf Spot of Cucumber

(*Pseudomonas lacrymans*, E. F. Smith and Bryan)

This disease is also uncommon on cucumbers in England, but it is widespread and destructive in the United States. It was first described by Smith and Bryan [15]. The disease is characterized by brown, angular spots on the leaves, which, in the early morning, show a bacterial exudate on the under surface. The spots dry up, and the central part falls away, leaving a hole. The disease may also produce spots on the fruit, from which a yellowish gummy substance exudes. The fruit spots may become invaded secondarily by other bacteria, causing a soft rot. Young plants, if badly attacked, may be seriously crippled in growth. Infection takes place through the stomata. According to Carsner [16] the bacteria are sometimes carried over in the micropyles of the seed.

In England, where the disease occurs only under glass, it can be controlled by dusting the plants with sulphur.

Black Rot of Cabbage and other Cruciferous Plants

(Pseudomonas campestris, (Pam.) E. F. Smith)

Pseudomonas campestris is rod-like, $0.7-3 \times 0.4-0.5 \mu$; cells single or in chains, young cells with a single polar flagellum; it is aerobic, nitrate-reducing, non-viscid, slowly liquefying gelatine; surface colonies on agar plates pale yellow, circular, or slightly irregular.

This disease attacks cultivated forms of the genus Brassica, particularly cabbage, cauliflower, and turnip. It is destructive in the United States, and is generally prevalent in Europe, although rather rare in Britain. The usual symptoms are a yellowing of the foliage accompanied by blackening of the veins. The disease is often followed by a soft rot induced by other bacteria. Seedlings, if attacked, may be killed.

Infection may take place through wounds in the leaves caused by mechanical agencies or by insects, and Smith[17] has shown that infection occurs commonly through the marginal water-pores of the leaves. The cotyledons of the cabbage may be infected through the ordinary stomata. The organism spreads rapidly in the vessels.

Walker and Tisdale[18] have pointed out that seed contamination is frequent. To avoid infection from this source Walker[19] advises disinfection by immersing the seed for thirty minutes in a 0.1 per cent. solution of corrosive sublimate or in water at a temperature of 122° F.

The disease is most prevalent in hot weather, and it can be avoided to some extent by growing susceptible crops during the cooler parts of the year.

Soft Rot of Carrot and other Plants

(Bacillus carotovorus, L. R. Jones)

Bacillus carotovorus is rod-shaped, cells single or catenulate, peritrichous; it is aerobic and facultatively anaerobic, gas-forming (with sugars), slowly liquefying gelatine; surface colonies on agar plates greyish-white or slightly yellow, round, raised, smooth, with a well-defined margin.

This is one of the most widespread bacterial diseases of plants, causing a soft rot of fleshy roots (carrot and turnip),

tubers (potato), rhizomes (Iris), stems (celery), bulbs (onion),
and fruits (tomato). It does not often attack green tissues,
but it commonly causes foot-rot of cucumbers and melons
grown under glass in England. The causative organism was
first described by L. R. Jones [20], but Potter's [21] *Pseudomonas
destructans*, recorded as causing a white rot of turnips, is
probably the same organism. Harding and Morse [21a] have
made a comparative study of *B. carotovorus* and certain closely
related forms of bacteria.

The bacterium usually enters the tissues through minute
wounds. Living chiefly in the intercellular spaces it poisons
the host protoplasm by the secretion of a toxin and disintegrates
the middle lamella of the cell walls.

Bewley [22] points out that the foot-rot of cucumbers caused
by this organism can be prevented by keeping the soil round
the stem relatively dry.

Soft Rot of Arum and other Plants

(Bacillus Aroideae, Townsend)

This organism is very closely related to *B. carotovorus*, but Massey [22a]
points out that it differs from *B. carotovorus* in pathogenicity and
in not producing gas during its action on dextrose, lactose, and
other sugars. *B. Aroideae* causes a soft rot of the corms of the Arum
Lily (Richardia or Calla), tomato fruits, cauliflower, and kohlrabi,
but it does not attack Iris rhizomes.

Bewley and Williams [22b] advise the following treatment for
affected Arum corms: at the end of the dormant period the corms
should be washed vigorously and all decayed portions removed
from them; the corms should then be steeped for seven hours in
a solution of corrosive sublimate at the rate of 1 oz. to 6 galls. of
water, after which the corms should be replanted in sterilized soil.

Crown Rot of Rhubarb

(Bacterium rhaponticum, Millard)

The most serious disease in the extensive rhubarb-growing area in
Yorkshire is that caused by *Bacterium rhaponticum*. It has been
studied by Millard [23], who states that the organism enters the crown
at about soil-level or through the lateral roots, and causes a soft
brown rot of the inner tissues. 'Crowns' affected in this way are
useless for forcing. The disease is cumulative owing to the practice,

prevalent until recently, of growing rhubarb on the same land for many years. The disease is spread through the soil by the incorporation of affected tissues. By careful attention to plant hygiene and by crop rotation it should be possible to reduce the disease considerably.

Blackleg of Potato

(*Bacillus phytophthorus*, Appel)

Bacillus phytophthorus is rod-like, $1.3–1.8 \times 0.9 \mu$; cells single or in chains, peritrichous; it is Gram-negative, nitrate-reducing, aerobic and facultatively anaerobic, liquefying gelatine; colonies on agar plates circular, greyish-white.

Other names for the organism are *B. melanogenes*, Pethybridge and Murphy, and *B. atrosepticus*, van Hall.

'Blackleg' is a widespread potato disease which has been investigated by Appel[23a], Pethybridge and Murphy[24], Kotilla and Coons[25], and others. It rarely causes serious damage in the growing crop, and is commoner in early than in late varieties. The disease is so named because the organism causes a soft black rot of the lower part of the stem, which is accompanied by premature yellowing of the foliage and curling upwards of the leaflets. The organism attacks the parenchymatous tissues, and lives chiefly in the intercellular spaces. It may pass along the stolons to the heel end of the tubers, which may become wholly or partly rotted. On cutting open a diseased tuber the affected tissues turn black upon access of air. The disease is chiefly transmitted by the seed tubers, which, if only slightly affected, are not easily detected. Shoots arising from such tubers become attacked and may be killed completely before emergence above the soil, or they may be affected later as previously described. Leach[26] states that infection of the shoots only readily occurs after the disappearance of the starch from the tuber; before this, the bacteria, on passing from the vascular tissue of the tuber into the parenchyma, are prevented from proceeding far by the formation of corkbarriers. The disease may perhaps also be contracted from the soil, and the tubers can be infected through the lenticels.

Pethybridge and Murphy[24] have called attention to the great losses sometimes caused by *B. phytophthorus* in potatoes stored in clamps or pits. Under these conditions the organism

spreads rapidly. Care should be taken to prevent the inclusion in the clamps of tubers affected by this bacterium, and the clamps should be adequately ventilated.

Leach [27] has traced an interesting connexion in Minnesota between this bacterium and the seed-corn maggot (*Phorbia fusciceps*). The bacterium is normally present in the intestinal tract, and the surface of the eggs is contaminated by it. The eggs are often laid near the seed tubers, which, on being attacked by the maggots, become infected by the bacterium.

Brown Rot of Solanaceae, &c.

(*Pseudomonas solanacearum*, E. F. Smith)

This organism causes a serious wilt of potatoes, tomatoes, tobacco, castor-oil, and other plants in the United States and India. It has been reported on tomatoes in Britain, but it is rarely harmful, as the temperature is usually too low for the organism to thrive. In diseased plants the vascular bundles and sometimes also the parenchyma are discoloured brown. Smith [28] states that infection occurs through wounds or through the stomata; potato plants are often invaded from the seed tubers. Ashby [29] has shown that this bacterium also causes a vascular wilt of bananas in the West Indies.

'Spraing' ('Sprain') of Potato Tubers

(*Pseudomonas solaniolens*, Paine)

Under the alternative name of 'Internal Rust Spot' Paine [30] states that this disease is caused by *P. solaniolens*. The disease appears usually in the form of brown spots in the flesh of potato tubers. The mode of infection is at present unknown. Atanasoff [30a] states that Sprain is of constant occurrence on certain soils rich in organic matter in Holland, and considers that the disease is probably caused by a parasitic organism. He points out that affected tubers sometimes show distinct scabbing, depressions, and malformations. Millard [30b] states that the disease has been successfully reproduced by inoculating tubers with a bacterium isolated from lesions of Internal Rust Spot.

Streak, Stripe, and Chocolate Spot

(*Bacillus Lathyri*, Manns and Taub.)

Bacillus Lathyri is rod-like, $1.2-2 \times 0.8-1 \mu$; cells usually single, rarely in pairs, peritrichous; it is aerobic, Gram-negative, nitrate-reducing, slowly liquefying gelatine; surface colonies on agar plates yellow, round, raised, wet-shining.

B. Lathyri was first found to be the cause of 'Streak' disease of sweet peas and clover by Manns and Taubenhaus [31]. It has since been claimed to be the cause of 'Stripe' of tomatoes by Paine and Bewley [32] and of 'Chocolate Spot' of beans (*Vicia faba*) by Paine and Lacey [33]. According to Manns and Taubenhaus [31] the disease is often carried over in the seed.

The most characteristic sign of the disease is the presence of elongated, brown streaks on the stems, but discoloured spots and blotches also occur on the leaves, especially in beans. Diseased tomato fruits show brown, sunken patches. The organism enters the host through small wounds often produced by insects, either above or below soil-level; it spreads chiefly in the cortex and pith, causing browning.

Young plants that are severely attacked may be killed, and in beans the plants may be defoliated, but, commonly, growth is not greatly impeded by the bacterium.

With tomatoes, there are considerable differences in suscepti- bility, 'Ailsa Craig' being very resistant. Generally speaking, a succulent type of growth favours the disease, while a hard form of growth tends to inhibit it. The disease can be much reduced in susceptible varieties by manuring them with sul- phate of potash at the rate of five cwt. per acre.

As indicated on page 29 Vanterpool and Dickson * consider that tomato 'Stripe' is caused by a mixed infection of two viruses.

As regards Chocolate Spot of beans it is doubtful whether all the brown lesions commonly seen are due to *B. lathyri*. Some of these brown areas may be caused by cold winds and frost in early spring, and some to aphis exudates.

Bacterial Disease of Peas

(*Pseudomonas seminum*, Cayley)

This disease of *Pisum sativum* is commonly transmitted through the seed, although infection may also occur from the soil. It has been investigated by Cayley [34]. Infected peas have a brown spot in the middle of each cotyledon. Such seeds may germinate normally, but the bacterium, on passing into the young shoot,

* For references see chapter on Virus Diseases.

sometimes kills the extremity; in other cases brown streaks appear on the stem. The leaves may become spotted with darkened veins. It is not known how the cotyledons become infected, but the organism has been found in the pods and in the funicles of the seeds.

According to Cayley [34] the organism forms spores in culture, an exceptional feature for bacteria pathogenic to plants.

A disease of pea seeds, known as 'Marsh Spot', of common occurrence in Britain, is also characterized by brown, necrotic spots in the cotyledons, but what connexion, if any, this disease has to the one investigated by Cayley is not yet known.

Yellow Disease of Hyacinths

(*Bacterium Hyacinthi*, Wakker)

Bacterium Hyacinthi is rod-like, $1.0–3.6 \times 0.4–1\ \mu$; cells single or in pairs, rarely in fours, with a single polar flagellum; it is aerobic, Gram-negative, non-nitrate reducing, slowly liquefying gelatine; surface colonies on agar plates yellow, flat, roundish, smooth, wet-shining.

This disease occurs commonly in Holland, where it has been investigated by Wakker [25]. It is also found in other countries on account of the importation of diseased bulbs from Holland. On cutting open longitudinally a diseased bulb, yellow streaks are seen. The bacterium is at first confined to the vascular bundles, but it spreads later to other parts, and it is then often accompanied by other bacteria and mites. If slightly diseased bulbs are planted they give rise to abortive shoots; badly affected bulbs often rot in the ground. The leaves are initially attacked from spring onwards and show yellowish-brown streaks. Infection often extends from the leaves down to the bulbs, and daughter-bulbs may be infected from the mother-bulb by way of the 'plateau' at the base.

Dutch growers attempt to control the disease by destroying plants which show the symptoms of disease in the leaves in spring. There are great differences in varietal susceptibility, and some of the most susceptible kinds are no longer cultivated. Van Slogteren [25a] recommends that, after harvesting, the bulbs should be kept for some time at a temperature of $95–100°$ F. in order to facilitate the development of the disease if present. In this way the disease is made recognizable, or,

failing this, progresses to such an extent that the bulbs fail to develop when planted.

Tobacco Wild Fire
(*Pseudomonas tabacum*, W. and F.)

In certain seasons this disease may cause an almost complete loss of crop. Seedlings may be killed outright by the bacterium, but the more usual kind of attack is on the leaves of bigger plants, in which large dead areas may be produced. The disease is often transmitted with the seed.

There are several other important bacterial diseases of tobacco, including Angular Leaf Spot (*Bacterium angulatum*, Fromme), and Black Fire (*Bacterium mellcum*, Johnson).

Angular Leaf Spot of Cotton
(*Pseudomonas malvacearum*, E. F. Smith)

This disease probably occurs wherever cotton is cultivated, and is sometimes serious. It has been investigated chiefly in the United States by E. F. Smith [36] and Faulwetter [37]. Infection takes place through the stomata. The leaf lesions first appear as translucent spots, which become brown and extend until bounded by the veins. Affected foliage may fall prematurely. The young bolls may also be attacked, when they may be shed or become rotten. The bacterium is often carried over with the seed, either on the surface or within.

Watermark Disease of the Cricket-bat Willow
(*Bacterium Salicis*, Day)

The cricket-bat willow (*Salix coerulea*) is often affected in the eastern counties of England by a die-back of the crown, which gradually leads to the death of the tree. Day [38], who has investigated the disease, considers that the primary pathogen is a bacterium, named *B. Salicis*, which enters the vascular system through minute wounds caused by insects. The organism lives chiefly in the vessels, and its presence there causes a wilt and browning of the leaves in the early summer. Wherever insects puncture the bark of dead branches, bacteria exude in colourless, sticky masses, which turn brown. The disease generally progresses until the entire crown is involved. On cutting across an affected branch the invaded wood is seen to be deeply stained; if the disease is in an early stage, watery masses of bacteria exude from the vessels, which give the name of 'watermark' to the disease.

As the branches die back, they become secondarily invaded by the fungus *Cytospora chrysosperma*, which may behave as a weak parasite.

E

According to Day the disease is most prevalent n wet subsoils and where the crowns of the trees are partially suppressed owing to overcrowding. He suggests that affected trees should be destroyed in the early spring before the bacteria exude, and that careful attention should be given to the soil and light requirements of the tree.

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CHAPTER V

DISEASES CAUSED BY ACTINOMYCETES

The Actinomycetes are a group of organisms which show relationships to some of the filamentous bacteria and also to certain fungi, but they are probably most nearly related to the former. They consist of exceedingly narrow, branched, sparsely septate threads, the diameter of which varies from 0·3 to 1·2 μ. It is doubtful whether the cells contain nuclei. Spore formation occurs by the breaking up, from the apex backwards, of the vegetative filaments into oidium-like bodies. Endospores are not formed. Like many bacteria the Actinomycetes are Gram-positive in staining reaction.

The Actinomycetes are widely distributed, being commonly found in soils; several species are pathogenic to man and the higher animals.

The group has been monographed by Lieske[1], and its morphology has been investigated by Drechsler[2].

Actinomyces, Harz

The characters of the genus are those of the group. The species can only be diagnosed adequately by their behaviour on standard culture media.

Actinomyces scabies, (Thaxter) Güssow — Common Potato Scab.

One of the forms causing Common Potato Scab was first described by Thaxter[3] under the name of *Oospora scabies,* and this was transferred later to the genus Actinomyces. Subsequently, Lutman and Cunningham[4] investigated a number of different kinds of Common Potato Scab, and considered that, notwithstanding certain differences in the external form of these 'scabs' and in the cultural reactions of the organisms isolated from them, all the strains should be grouped together in the one species, *A. chromogenus,* Gasparini. More recently, Wollenweber[5], and Millard and Burr[6], in comparative investigations of different forms of Actinomyces, have concluded that the various types of Common Potato Scab are caused by different species of Actinomyces, of which one is now named *A. scabies,* (Thaxter) Güssow emend. Millard and Burr. This

species produces brown nodular outgrowths on potato roots and stolons as well as scabs on the tubers. For a full diagnosis of the different kinds of scab and of the causative organisms the reader is referred to the paper by Millard and Burr.

The following chief types of Common Potato Scab are differentiated by Millard and Burr [6]:

1. *Superficial scab.*
   This is a brownish abrasion of the skin of the tuber, usually only slight in extent.

2. *Ordinary scab.*
   This is characterized by an irregularly concentric series of wrinkled layers of cork arranged around a central core or depression.

3. *Pitted scab.*
   This is marked by a depression in the tuber, bordered by the ragged edges of the torn skin. In the more virulent forms the depressions may be 3-4 mm. deep, and deep furrows may be formed where two or more scabs coalesce. This is the type of scab originally described by Thaxter.

4. *Stud scab.*
   Here there is a distinct swelling 2-3 mm. above the surface.

5. *Tumulus scab.*
   Like the preceding, but the sides of the swelling are sloping instead of vertical.

Generally speaking, the type of scab on any one tuber is constant, and the type may be constant throughout the whole crop, because the environmental conditions for any one crop will be practically uniform and will tend to favour one species of Actinomyces rather than several. The variety of tuber has only a slight influence in modifying the type of scab.

Infection of the tubers takes place through the lenticels. The disease first appears in the form of small brown spots, which increase in size to a diameter of about 4 mm. As the spots extend, the skin ruptures and a 'scab' is formed. During invasion of the surface cells by the parasite the host reacts by the formation of cork layers, so that the scabs are more or less corky on freshly-lifted tubers. Scab spots are often covered with the delicate white threads of the Actinomyces, but these quickly shrivel on exposure. Two or more scabs may coalesce,

and, in bad attacks, the whole of the tuber may be covered with scabs. In rare instances the tubers may be so badly scabbed as to be unsaleable, but a slight attack does not detract seriously from the value of the crop.

Common Scab occurs most frequently on light, sandy, or gravelly soils, and is most prevalent in dry seasons. It is rare on peaty and heavy soils. Millard[7] considers that the hydrogen-ion concentration of the soil is not a direct factor in determining the incidence of Scab. He has shown that in soils heavily infected with Actinomyces organisms the disease can often be controlled by green-manuring. On small areas this can be done by incorporating grass-cuttings, &c., into the soil before the potatoes are planted, but, on larger areas, ploughing in a crop such as mustard or rye is necessary. Millard and Taylor[8] consider that green-manuring favours the growth of saprophytic forms of Actinomyces and bacteria, thus leading to the suppression of the scab organisms.

Actinomyces tumuli, Millard and Beeley   Mangold and Beet Scab.

This species has been isolated by Millard and Beeley[9] from the raised type of scab commonly found on mangolds and beet, Such scabs are mound- or knob-like protuberances, measuring up to 20 mm. across and rising 6 mm. above the surface. In these raised scabs the outer pericyclic cells proliferate actively in response to invasion by the parasite, and do not form a cork cambium until at least a month later.

A pitted type of scab also occurs on mangolds and beet, caused, according to Millard and Beeley[9], by *A. scabies*, (Thax.) Güssow, emend. M. and B. As in the potato, this form also produces nodular outgrowths on the true roots.

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CHAPTER VI

DISEASES CAUSED BY MYXOMYCETES

THE Myxomycetes or Mycetozoa are a curious group of organisms devoid of chlorophyll, having a preponderance of plant characters, but possessing some animal characters. They are related on the one hand to the lower Fungi, and, on the other, to certain groups of Protozoa.

On germination the spores give rise to naked masses of protoplasm, sometimes provided with a single cilium, by means of which movement in water may be effected. These swarm spores become amoeboid, and grow or fuse with one another to form a plasmodium, which may creep slowly from place to place. After further growth, the plasmodium divides to form a large number of minute spores, which are sometimes formed in structures of macroscopic size termed 'sporangia'. The plasmodia and 'sporangia' of some species are brightly coloured.

The great majority of the Myxomycetes live on rotten wood or other plant debris, but a few, included in the family Plasmodiophoraceae, are parasitic on plants. The Plasmodiophoraceae are sometimes separated from the Myxomycetes and placed in a group of their own.

Plasmodiophora, Woronin

Spores spherical, free from one another, completely or almost completely filling the host cells.

Plasmodiophora Brassicae, Wor.   Finger-and-toe, Club Root, Anbury.

Spores spherical, colourless, $1 \cdot 6 – 3 \cdot 3 \, \mu$ in diameter, each forming an amoeba-like body with a single cilium on germination ; parasitic in the roots of cruciferous plants, causing extensive hypertrophy.

Finger-and-toe disease is prevalent wherever cruciferous crops such as turnips, swedes, Brassicae, radishes, &c., are grown upon acid soils which have a high water-content when the host plants are young. The disease is characterized by the presence of large galls on the roots, which may lead to impoverishment of the plants or render the roots worthless. Upon disintegration of the galls the spores pass into the soil, where they may retain their vitality for several years.

The disease was first investigated in detail by Woronin[1]. It has been studied more recently by Nawaschin[2], Chupp[3], Kunkel[4], and others.
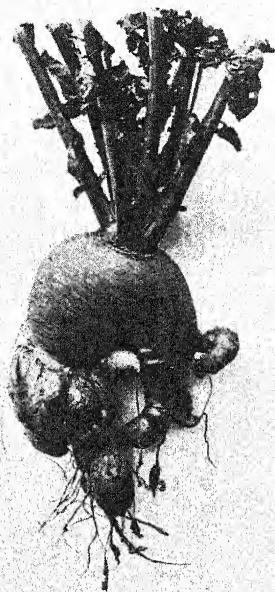
Fig. 4. 'Finger-and-toe' of turnip. ½ natural size. (*A. Smith.*)

On germination the spore forms an amoeboid body with a single cilium, which moves about to a limited extent in the soil water. The initiation of infection of the host is not clearly understood, but, apparently, the root hairs and other superficial cells of the root can be penetrated by the swarm spores or by the 'amoebae' into which the swarm spores are quickly transformed. Once within the host, the 'amoeba' grows rapidly into a plasmodium, which penetrates the cell walls and becomes divided into daughter-plasmodia. A single cell may

finally contain several plasmodia.   The plasmodia may pene-
trate as far as the cambium.   Both the infected cells and
others near them become enlarged and divide profusely so that
large galls are formed.   The plasmodia divide up to form spores,
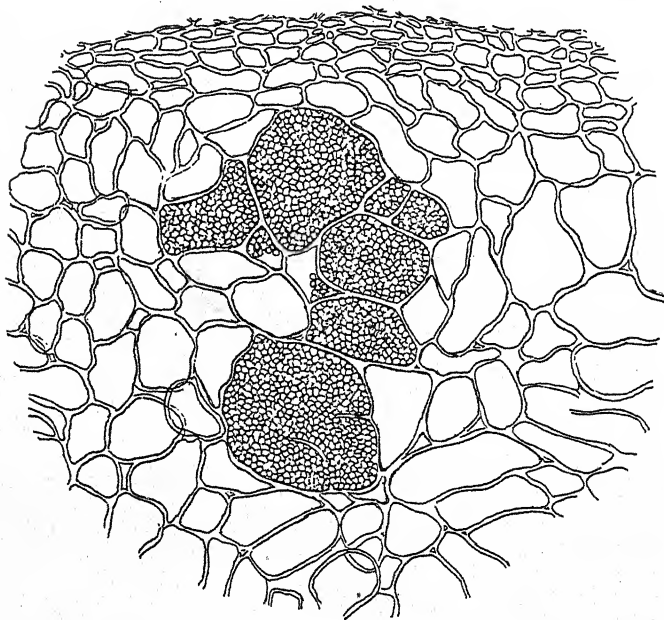
FIG. 5. *Plasmodiophora Brassicae,* spores in cells of *Sisymbrium officinale.* × 350.
(*R. W. Marsh.*)

which often completely fill the cell cavity.   Old galls break
open, and in this way other micro-organisms enter and cause
a soft rot, which allows the spores to pass into the soil.   If
galled roots are fed to animals the spores may be disseminated
with the dung, owing to the probable inclusion of uneaten
portions ; the spores are apparently killed by passage through
the alimentary canal.

Generally speaking, only young plants can be infected, and
the greater the water-content of the soil the more likely is
infection to be brought about.

Owing to the tendency of this disease to appear in acid soils
it can be checked appreciably by the addition of a heavy

dressing of lime—up to four tons per acre, which should be applied before planting the crop preceding the susceptible one. As a high water-content of the soil is necessary for infection, cruciferous crops should only be grown on well-drained land. In some districts where the disease is prevalent, the period in the rotation between two susceptible crops is extended : thus in Scotland temporary grass leys are often retained longer than usual between two successive turnip or swede crops. As the parasite attacks certain cruciferous weeds such as charlock, these should be eradicated.

With turnips, swedes, and radishes there are considerable differences in varietal susceptibility, but fuller information is required about this in Britain. The swede 'Studsgaard Bangholm' appears to be highly resistant. Fast-growing varieties of turnips and swedes are less susceptible than slow-growing ones.

In seed-beds of Brassicae the disease can be prevented by watering the soil with weak corrosive sublimate, 1 oz. in 10 gallons of water.

Spongospora, Brunchorst

Spores aggregated in hollow, ball-like masses.

Spongospora subterranea, (Wallr.) Lagerheim Powdery or Corky Scab of Potatoes.

Spore-balls irregularly globose or elliptical, hollow, brownish, 40–50 μ in diameter ; individual spores spherical, 3–4 μ in diameter ; parasitic on the underground parts of the potato.

Powdery Scab is probably indigenous in Peru and Ecuador, and it is widespread in countries where potatoes are now cultivated. It usually occurs only where the soil temperature is comparatively low and the moisture-content high. In Britain the disease is commoner and more destructive in the north than in the south.

S. subterranea attacks only the subterranean parts of the potato. On the tubers the symptoms are very diverse. The commonest form of the disease on these is the presence of 'scabs' about 6 mm. across and circular in outline ; such scabs

are raised when young, but are depressed when the skin has
broken away, leaving a brown, powdery mass of spore-balls.
Occasionally a raised scab continues to proliferate, and pro-
duces a gall-like excrescence which can only be distinguished
with difficulty from Wart Disease.   Another common form of
the disease in wet soils is 'canker', in which a large part of the
tuber is destroyed, leaving a deep depression.   The parasite
may also produce small galls on the roots, stolons, and parts of
the stems below ground.
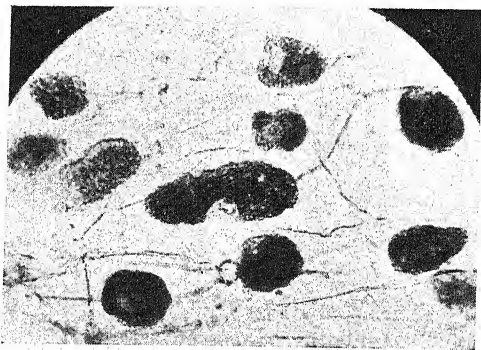
FIG. 6. *Spongospora subterranea*, spore-balls in cells of potato tuber. × 350.
(*G. H. Pethybridge.*)

Powdery Scab has been investigated by Brunchorst[5], who
first assigned the parasite to its correct systematic position,
and by Johnson[6], Osborn[7], Kunkel[8], and Melhus and Rosen-
baum[9].   The last-named authors succeeded in infecting the
tomato and certain other species of Solanum.

The spore-balls may remain alive in the soil for several years.
On germination each spore produces an 'amoeba', which has
a limited power of movement in the soil water.   Under favour-
able conditions several 'amoebae' coalesce to form a plas-
modium, which, according to Kunkel[8], is the infecting unit.
Under drier soil conditions Kunkel states that the 'amoebae'
may encyst and remain dormant until the water-content
increases.   The initiation of infection of the tuber is not clearly
understood, but the plasmodium soon passes into the tissues
below the skin.   The plasmodium branches, and parts of it

spread between the cells, which are ultimately penetrated. Some of the infected cells increase in size and divide rapidly, leading to the formation of the raised 'scabs' typical of young lesions.   The plasmodia are ultimately converted into spore-balls, and, shortly after, these may become exposed owing to the rupture of the host cell walls.   In the commonest type of Powdery Scab the parasite does not penetrate deeply into the tuber owing to the formation of a cork-barrier, but in the 'canker' form of the disease the plasmodia may extend throughout the thickness of the tuber. The lesions of Powdery Scab may be secondarily invaded by tuber-rotting micro-organisms.

Powdery Scab is most destructive in the 'canker' form. Where this type of the disease occurs, potatoes should not be grown on infected land for several years, and the drainage should be improved if possible. Tubers infected in any way by the parasite should not be used for seed.   In Britain there are no varieties of potatoes known to be immune from this disease.

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FUNGI AND THEIR CLASSIFICATION

THE Fungi constitute an immense group of lowly plant organisms. They are devoid of chlorophyll and obtain their carbon from organic compounds, living therefore as saprophytes or parasites. Many parasitic fungi cause serious diseases of crop plants.

The plant body of a fungus consists either of a single spherical cell, or, much more commonly, of branching threads or *hyphae*, collectively termed the *mycelium*. The hyphae, which are walled, may have cross-septa, but longitudinal divisions practically never occur in them. Growth in length of the hyphae takes place at the apex. In certain of the higher fungi part of the mycelium may become densely aggregated to form *sclerotia*. These are hard bodies, usually of considerable size, which undergo a period of rest before developing further. In other fungi the mycelium produces long, firm strands or *rhizomorphs*, by means of which the fungus grows from one place to another. The vegetative part of a fungus is usually embedded in the tissues of the host or other substratum, and only the reproductive part is visible, although in the true mildews the mycelium is mainly superficial. The mycelium of parasitic fungi may be intercellular or intracellular; in that of the former the hyphae often give rise to sucker-like processes or *haustoria*, which penetrate the host cells and withdraw food from them.

The reproductive units of the Fungi are bodies of microscopic size, termed *spores*, which are formed in many different ways. In the lower fungi many spores may be formed by the division of the protoplasm of a vesicle or *sporangium*; such spores may be naked masses of protoplasm provided with one or more cilia and capable of movement in water (*zoospores*), or they may be walled. Spores abstricted from hyphae are called *conidia*, and thick-walled spores formed within hyphae or other spores are termed *chlamydospores*. In the lower fungi thick-walled resting spores (*oospores* or *zygospores*) may be formed sexually. In the Ascomycetes the characteristic spore-forming structure is the *ascus*, a sac-like body, in which, when young, there is a nuclear fusion, and within which a definite number of spores, usually eight, is formed. In the Basidiomycetes the characteristic spore-producing structure is the *basidium*, a short hypha or sac-like body in which, or in the spore from which it arises, there is a nuclear fusion when young, and from which a definite number of spores, usually four, is abstricted. Asci and basidia are often aggregated together and surrounded wholly or

partially by sterile hyphae to form fruit-bodies, which may be large in size. A layer of asci or basidia, often interspersed with sterile cells (*paraphyses*), is termed an *hymenium*. Ascus fruit-bodies are sometimes embedded in a compact mass of mycelium called a *stroma*.

Wind is the most potent agent in the dissemination of many fungi, the spores being so light that they are carried considerable distances in a living condition. The spores of some fungi are shot forth violently into the air at maturity. Movements of water and insects also play a part in the dispersal of some fungi. Many of these organisms are disseminated by contact with, or inclusion in, materials which are moved from place to place ; fungi pathogenic to plants are often seed-borne, or are carried in tubers, bulbs, and corms.

Parasitic fungi invade plant tissues in a variety of ways. Zoospores having come to rest on the surface of the host may penetrate the epidermal cells directly, but much more commonly the spore germinates on the surface and emits a germ-tube, which either enters the tissues by way of a stoma or lenticel, or which penetrates an epidermal cell directly. Many dangerous fungi can infect their hosts only through wounds, or by first developing upon dead tissues. As indicated in Chapter I, environmental conditions play a very important part in determining whether infection takes place or not. Corky tissues cannot generally be penetrated by parasitic fungi. Fungi which cannot be cultivated on artificial media are termed *obligate parasites*.

A parasitic fungus may pass the whole of its life-cycle on a single host (or upon several distinct host species), when it is said to be *autoecious*. Another kind of parasitic fungus, however, may pass part of its life-cycle on one host or group of host plants, and another part on a different host or group of host plants, as in certain Rust Fungi, when it is said to be *heteroecious*.

Distinct physiological varieties, differing in host relationships, but essentially the same morphologically, may exist within the limits of a single species. Thus Yellow Rust (*Puccinia glumarum*) occurs on wheat and barley, but the variety of Yellow Rust on wheat cannot normally infect barley, or vice versa. Such physiological varieties are frequently termed *biologic* or *specialized forms*.

For complete diagnoses of the genera and species of parasitic fungi the reader should consult Saccardo's *Sylloge Fungorum* or Rabenhorst's *Kryptogamen Flora*. For descriptions of the chief genera the volumes of Engler and Prantl's *Pflanzenfamilien* on fungi will be found useful. Books dealing with the morphology and mode of life of fungi are de Bary's *Comparative Morphology and Physiology of the Fungi*, *Mycetozoa and Bacteria*, Gwynne-Vaughan's *Fungi*, Gäumann's *Vergleichende Morphologie der Pilze*, and Gwynne-Vaughan and Barnes's *Structure and Development of the Fungi*.

The classification of fungi is based upon the methods of spore formation. The following is a synopsis of the scheme of classification used in this book, groups containing pathogenic forms alone being included :

A. PHYCOMYCETES

Spores usually formed in sporangia at some stage in the life-cycle.

1. Mycelium absent or consisting of very slender hyphae ; reproduction chiefly by zoospores . . . CHYTRIDIALES
2. Mycelium profuse and consisting of comparatively wide hyphae ; asexual reproduction by zoospores formed in elongated sporangia ; several egg-cells in an oogonium . SAPROLEGNIALES
3. Mycelium as in (2) ; asexual reproduction by zoospores formed in globose sporangia, or by conidia ; one egg-cell in an oogonium . . . . . PERONOSPORALES
4. Mycelium as in (2) ; asexual reproduction by non-motile spores ; resting zygospores (zygotes) formed by fusion of undifferentiated or only slightly differentiated gametes . MUCORALES.

The Saprolegniales and the Peronosporales are included in the Oomycetes, and the Mucorales in the Zygomycetes.

B. ASCOMYCETES

Mycelium profuse and generally much septate ; spores formed in asci at some stage in the life-cycle.

1. Asci formed in a group in small, spherical, closed perithecia . . . . . . ERYSIPHALES
2. Asci formed in a palisade-like manner under the cuticle or epidermis of leaves, stems, or fruits. . EXOASCALES
3. Asci scattered in small, closed, spherical cleistocarps, or not enclosed in a definite fruit-body . . PLECTASCALES
4. Asci formed in cup-like or disk-like apothecia PEZIZALES
5. Asci spread over the surface of large, irregularly-shaped fruit-bodies . . . . . . HELVELLALES
6. Asci formed in small apothecia which open irregularly . PHACIDIALES
7. Asci formed in small apothecia which open by a longitudinal split . . . . . HYSTERIALES

The Pezizales, Helvellales, Phacidiales, and Hysteriales comprise the Discomycetes.

8. Asci formed in small perithecia which open by means of an ostiole ; perithecia brightly coloured and somewhat soft . . . . HYPOCREALES

9. Asci formed in ostiolate perithecia, which are embedded
   in a hard, black stroma ; perithecial walls not clearly
   delimited from the stroma .   .   .   .   DOTHIDIALES

10. Asci formed in dark-coloured, ostiolate perithecia, which
    have distinct walls even when embedded in a stroma   .
                                                    SPHAERIALES

   The Hypocreales, Dothidiales, and Sphaeriales comprise the
Pyrenomycetes.

C. BASIDIOMYCETES

Mycelium profuse and usually much septate ; spores formed on
basidia at some stage in the life-cycle.

1. Most of the mycelium converted into brand-spores at
   maturity, which produce basidia after a period of rest   .
                                                    USTILAGINALES

2. Basidia arising from or in teleutospores, which are formed
   in scattered pustules from a part of the mycelium ; basidia
   transversely septate   .   .   .   .   UREDINALES

3. Basidia formed directly by the mycelium, transversely
   septate, arranged in a definite hymenium   AURICULARIALES

4. Basidia formed directly by the mycelium, non-septate.
   arranged in an ill-defined hymenium on the surface of
   leaves, &c.   .   .   .   .   .   .   EXOBASIDIALES

5. Basidia as in (4), arranged in an hymenium which is
   exposed throughout development and is spread over a
   smooth surface, teeth, anastomosing gills, or pores .   .
                                                    APHYLLOPHORALES

   (a) Fruit-body erect, fleshy, often branched   *Clavariaceae*

   (b) Fruit-body resupinate or bracket-like, tough ; hy·
       menium spread over a smooth or rugose surface .
                                                    Thelephoraceae

   (c) Fruit-body resupinate or pileate, fleshy ; hymenium
       spread over spines, granules, or warts   .   *Hydnaceae*

   (d) Fruit-body resupinate or bracket-like, fleshy ; hy·
       menium smooth, or spread over veins, or anasto-
       mosing pores .   .   .   .   .   .   *Meruliaceae*

   (e) Fruit-body bracket-like, fleshy ; hymenium lining
       free and separate tubes .   .   .   *Fistulinaceae*

   (f) Fruit-body resupinate or bracket-like, tough ; hy·
       menium lining tubes, or covering gills or teeth,
       homogeneous with the substance of the pileus   .
                                                    Polystictaceae

   (g) Fruit-body as in (f); hymenium lining tubes
       which form a layer distinct from the substance of
       the pileus   .   .   .   .   .   .   *Polyporaceae*

6. Basidia as in (4); hymenium at first covered by a volva or ring, becoming exposed at maturity and spread generally over the surface of gills . . . AGARICALES

The Auriculariales, Exobasidiales, Aphyllophorales, and Agaricales are a part of the Hymenomycetes.

D. DEUTEROMYCETES (*Fungi Imperfecti*)

Spores formed chiefly on conidiophores, there being no sporangia, asci, or basidia in the life-cycle so far as is known at present.

1. Spores formed in pycnidia at some stage in the life-cycle . . . . SPHAEROPSIDALES

2. Conidiophores densely aggregated in pustules MELANCONIALES

3. Conidiophores generally separate from each other . . .
HYPHOMYCETES

4. No spore stages at present known . . MYCELIA STERILIA

FUNGUS DISEASES: CHYTRIDIALES, SAPROLEGNI-ALES, PERONOSPORALES, MUCORALES

CHYTRIDIALES

MYCELIUM absent or consisting of very slender hyphae. Repro-duction by motile cells, formed in sporangia, which may be thin-walled, liberating these cells directly, or thick-walled, having a prolonged resting period before germination.

Synchytrium, de Bary and Woronin

Mycelium absent. The plant body develops either into a 'sorus' of thin-walled sporangia which liberate zoospores or motile gametes directly, or into thick-walled sporangia (often mis-called 'spores') in which zoospores are formed after a period of rest.

Synchytrium endobioticum, (Schilb.) Perc. Wart Disease of Potatoes.

Resting sporangia thick-walled, brownish-black, about $52\,\mu$ in diameter. On germination the resting sporangia produce numerous uniciliate zoospores, which, upon infecting the host, form sori of thin-walled sporangia. The latter directly produce zoospores or motile gametes which fuse in pairs. Infection by the product of two fused gametes results in the formation of a resting sporangium, and by a single zoospore, of thin-walled sporangia.

This is one of the most serious diseases of the potato. It was first briefly described by Schilberszky[1] in Hungary, and, more recently, it has been investigated by Johnson[1a], Percival[2], Curtis[3], Köhler[4], and Glynne[5]. During recent years it has become widespread, and it now occurs in most European countries, the United States, Newfoundland, and South Africa. In Great Britain important potato-growing areas in Scotland, Lincolnshire, and Cambridgeshire are, however, still practically free from the disease. The fungus also infects tomatoes, whilst *Solanum nigrum, S. dulcamara, S. alatum,* and *Hyoscyamus niger* have been found to become slightly affected when planted in contaminated soil.

The disease is characterized by the presence of large or small warty excrescences on the tubers and rarely on the leaves; otherwise the growth of the potato plant is not affected. Warts do not arise on the roots of this plant. Infection of the young tubers takes place through penetration of
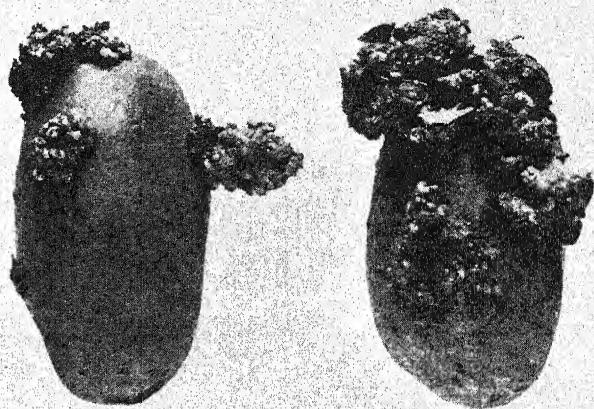
FIG. 7. *Synchytrium endobioticum*, warts on potato tubers.   Natural size.
(*G. H. Pethybridge.*)

the epidermis of the 'eyes' by the motile cells of the fungus. The host tissue in the vicinity of an infected cell proliferates abnormally, and in consequence of successive infections a wart of varying size and with a more or less convoluted surface is formed.   Sometimes practically the whole of the tuber is transformed into hypertrophied tissue, which then looks more like a dirty piece of cauliflower than a potato.   At first innumerable thin-walled sporangia are produced in the warts, but later in the season resting sporangia are formed.   Ultimately the warts may become invaded secondarily by other microorganisms, which cause a soft rot. Upon disintegration of the warts the resting sporangia pass into the soil, where they may

retain their vitality for several years and be a source of infection season after season. Köhler[4] states that the stolons also may be invaded, but infection of these organs does not result in the formation of warts. Weiss[6] has found that infection occurs within a wide range of temperatures, but only when the soil is very moist.
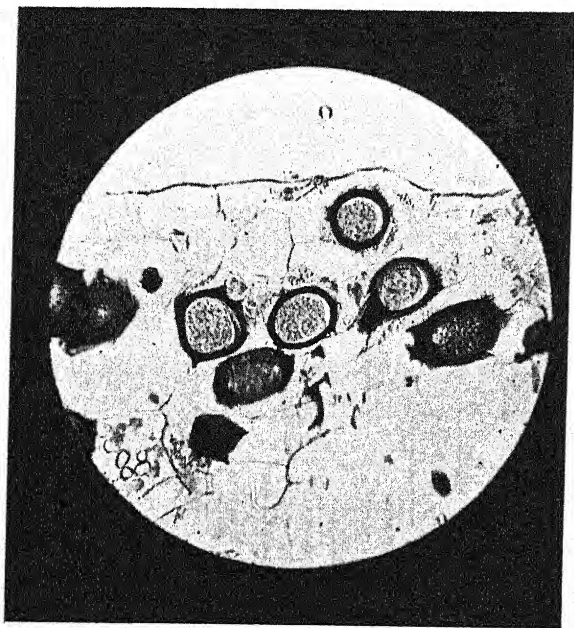
FIG. 8. *Synchytrium endobioticum*, resting sporangia in cells of wart. × 400.
(*G. H. Pethybridge.*)

Some varieties of potatoes and tomatoes are immune from the disease, and, as far as is known, this immunity remains constant under all conditions. Glynne[7], however, has shown that, under laboratory conditions, small protuberances bearing thin-walled sporangia may be formed on tubers of a few varieties which, from their behaviour in field trials and as a result of naked-eye inspection, were previously thought to be immune. According to Salaman and Lesley[8], immunity from wart disease is dependent upon one or more genetic factors.

There are, however, grades of susceptibility, some varieties of potatoes being much more susceptible than others.

The following varieties of potatoes commonly grown in the British Isles are immune from the disease :

Early varieties : ' Edzell Blue ', ' Snowdrop ', ' Immune Ash-leaf '.

Maincrop varieties : ' Great Scot ', ' Golden Wonder ', ' Abundance ', ' Ally ', ' Majestic ', ' Kerr's Pink ', ' Arran Comrade ', ' Duke of Perth ', ' Incomer ,' ' Arran Banner '.

As no satisfactory method of destroying the resting sporangia in the soil has yet been discovered, immune varieties only should be planted in infested ground.   In Great Britain the disease is notifiable to the Ministry of Agriculture, and, by the terms of the Wart Disease of Potatoes Order of 1923, the planting of susceptible varieties in infected soil is forbidden ; in view also of the danger of resting sporangia adhering to particles of soil on the tubers, immune varieties grown in infested soil are not allowed to be used as seed in non-infected areas.   Warty tubers should be burnt or should be well boiled first if fed to pigs.

Synchytrium Vaccinii, Thomas, causes small, red galls on young shoots and fruits of the cranberry and related plants.

Olpidium, Braun

Mycelium absent. The plant body forms a single zoosporangium which, upon dehiscence, penetrates the host by means of a tube through which the uniciliate zoospores or gametes emerge. Resting sporangia also occur.

Olpidium Brassicae, (Wor.) Dang.

This fungus attacks and kills the underground parts of seedling cabbages, cauliflowers, &c., especially when these are grown crowded together under very wet conditions.   According to Van der Meer [9] the zoospores infect the host only by way of the root hairs.   The effect of the fungus is sometimes closely similar to that of a ' damping off ' parasite, but the host may recover from attack by the development of new roots.   The resting sporangia are more or less star-shaped owing to

wrinkling of the outer coat, and are formed, according to Němec[9a], by the fusion of gametes in pairs.

Urophlyctis, Schröter

Mycelium exceedingly fine, producing numerous sporangia in the host tissues. These resting sporangia give rise to uniciliate zoospores on germination.
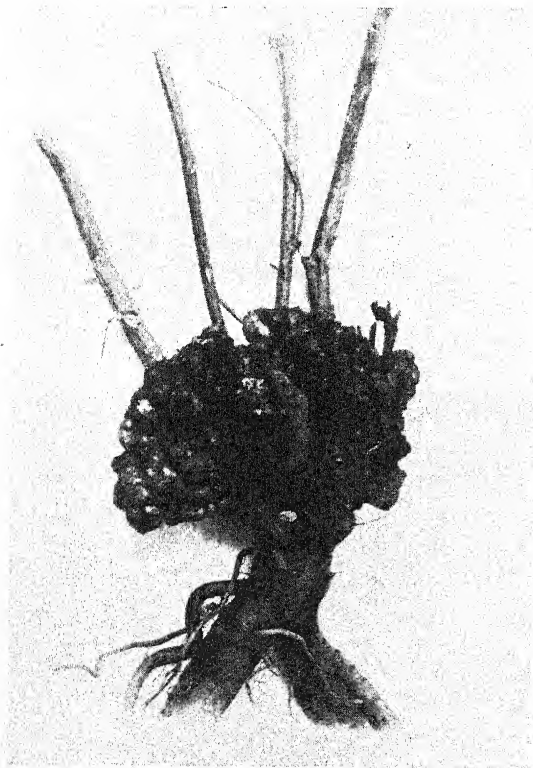
Fig. 9. 'Crown wart' of lucerne. $\frac{1}{2}$ natural size. (*A. Smith.*)

Urophlyctis Alfalfae, (Lagerh.) Magnus   Crown Wart of Lucerne (Alfalfa).

Resting sporangia brown, sub-spherical, slightly flattened on one side, $32-40 \times 28-32 \mu$.

The life-history has been investigated by Jones and

Drechsler [10]. This fungus occurs not uncommonly on lucerne
in England and in America. It causes the formation of large
galls on the root-stock at about soil-level, with the result that
the length of life of the ley is shortened. Affected plants often
wilt in hot weather.

Infection is brought about in spring through penetration of
buds at about soil-level by zoospores; an exceedingly fine
mycelium is first produced which grows through the tissues
and induces abnormal cell development in the parts attacked.
The mycelium rapidly forms 'turbinate' cells, which produce
both sporangia and other fine hyphae that in turn form other
'turbinate' cells, sporangia, and hyphae. The central part of
the invaded tissue becomes transformed chiefly into masses
of sporangia, which give a brown, mottled appearance to the
interior of the gall. Ultimately the gall becomes rotten
through invasion by other micro-organisms, and the resting
sporangia pass into the soil, where they may retain their
vitality for several years.

The disease is most prevalent on irrigated land or on land
that is liable to surface flooding in spring. Only well-drained
land should be used for growing lucerne. An extension of the
rotation is advisable on affected land.

Urophlyctis leproides, (Sacc. and Trab.) Magnus   Beetroot
Tumour.

Resting sporangia brown, sub-globose, depressed on one side, 45–
50 × 30 μ.

Roots of sugar-beet attacked by this fungus exhibit large
tumour-like growths in which the resting sporangia are em-
bedded. The disease has been recorded from north Africa and
western Europe. It must not be confused with 'crown-gall'
caused by *Bacterium tumefaciens*, which it resembles super-
ficially.

Physoderma, Wallroth

Hyphae narrow. Resting sporangia producing uniciliate zoospores
on germination.

Physoderma Zeae-maydis, Shaw

Resting sporangia smooth, brown, thick-walled, $20-30 \times 18-24\,\mu$, slightly flattened on one side, which is provided with a small cap.

This fungus occurs on maize in India and the United States, where Tisdale[11] states that it causes considerable damage. The parasite affects the leaves and stems, chiefly in the lower part of the plant, causing innumerable minute, brown spots. The leaves may be killed, and severe attack on the stems may cause 'lodging'. The brown spots contain the resting sporangia, which pass into the soil on the disintegration of the shoots. The sporangia are carried from the soil to the shoots by wind or through splashing by rain, germinating there to form zoospores which bring about infection.

SAPROLEGNIALES

Mycelium well developed. Asexual reproduction by means of biciliate zoospores formed in long, cylindrical sporangia. Resting spores produced by the fertilization or parthenogenetic development of egg cells enclosed in oogonia.

Aphanomyces, de Bary

Zoosporangia long and narrow, forming spores devoid of cilia at the moment of liberation. Resting oospores occur.

Aphanomyces euteiches, Drechsler

Oogonia sub-spherical, $25-35\,\mu$ in diameter, inner surface of wall sinuous; antheridia often branched; oospores sub-spherical or nearly ellipsoidal, $18-25\,\mu$ in diameter.

Jones and Drechsler[12] have investigated a serious root disease of peas caused by this fungus in the United States. It has recently been found in England on the same host.

The outer parts of the roots are penetrated by zoospores; the mycelium arising from these kills the host cells and forms oospores therein. Young plants may be killed, and older plants may wilt or be merely dwarfed. Secondary organisms frequently play a part in the destruction of the plants by penetrating the vascular tissues.

The disease is most prevalent on land of high moisture-content, hence such land should be avoided for growing peas. There are differences in varietal susceptibility, but oospores are formed in the outer parts of the roots even in resistant varieties.

Aphanomyces laevis, de Bary

As in *A. euteiches*, but the inner surface of the wall of the oogonium is not sinuous.

Peters[12] has shown that this species kills young sugar-beets in Germany. The life-history is essentially the same as in the previous species.

PERONOSPORALES

Mycelium richly developed. Asexual reproduction by the formation of spherical, pear-shaped, or oval sporangia or conidia, which may produce bi-ciliate zoospores or a germ-tube on germination. Resting oospores formed by the fertilization of an egg cell within an oogonium.

Pythium, Pringsheim

Zoospores liberated in an imperfectly differentiated state into a vesicle in which their formation is completed. Conidia often present, usually formed singly at the ends of hyphae. Oospores commonly found, the antheridia being always paragynous. The genus has been partly monographed by Butler[13].

Pythium de Baryanum, Hesse

Sporangia or conidia subspherical, $20-25\,\mu$; oospores globose, hyaline, smooth, $15-18\,\mu$.

This fungus is commonly found in soil and is one of the several causes of the 'damping-off' of seedlings, especially when grown closely together under very wet conditions. The fungus persists in the soil in the form of oospores. On germination, the oospores form zoospores or hyphae, which, on coming into contact with the seedling at about soil level, penetrate it. The host cells are killed immediately the fungus reaches them, with the result that the seedling falls over. Sporangia (or conidia) and oospores are formed in and upon the rotten tissues, the oospores ultimately passing into the soil. Where seedlings are grown under good conditions the

cuticle is better developed and often resists penetration by the fungus.

Braun [14] has shown that this fungus and related species of Pythium cause a stem rot of Pelargonium cuttings, which starts at the cut end and causes a blackening and shrivelling of the stem. Attack of this kind can be obviated by 'striking' the cuttings in clean sand.

According to Hawkins [15], *P. de Baryanum* also causes a rot of potato tubers in storage in California, where the disease is known as 'leak'. In this disease Hawkins and Harvey [16] consider that the fungus possibly penetrates the cell walls solely by mechanical pressure; it secretes a toxin, and perhaps also an enzyme, which dissolves the middle lamella.

Lehman [17] states that a rot of the fruits of *Capsicum annuum* var. *grossum* is due to *P. de Baryanum*; decay commences at the blossom end of fruits near soil-level.

Pythium aphanidermatum, (Edson) Fitzpatrick

Sporangia formed in large numbers as lateral, bud-like processes.

McRae [18] has investigated a soft rot of the rhizome of the ginger plant caused by this fungus, described under the name of *Pythium gracile*, Schenk. The first sign of the disease is a yellowing and wilting of the leafy shoots due to infection of the rhizome from oospores in the soil. The disease may be disseminated by using slightly affected portions of rhizomes for propagation. Affected plants should be burnt, and land should not be used for growing ginger oftener than once in five years. The disease is least prevalent on well-drained, sandy loams. This species also causes a serious stem rot of tobacco in Nyassaland.

Edson [19] has described the same fungus, under the name of *Rheosporangium aphanidermatum*, as the cause of a disease of sugar-beet in the United States. Carpenter [20] associates it with a destructive root rot of sugar-cane in Hawaii. Drechsler [21] has also found this fungus causing a serious rot ('cottony leak') of mature cucumbers in the United States, a striking feature of the disease being the outgrowth of a profuse white mycelium from the fruit.

Pythiacystis, R. and E. Smith

The sporangiophores bear numerous zoosporangia formed sympodially in clusters. Oospores unknown.

Leonian [22] suggests that this genus should be combined with Phytophthora.

Pythiacystis citrophthora, R. and E. Smith   Brown Rot of Lemons.

Zoosporangia ovate or lemon-shaped, papillate, $30-90 \times 20-60 \mu$, average $50 \times 35 \mu$, borne sympodially. Zoospores $10-16 \mu$ in diameter, two-ciliate.

This fungus was first described by Smith and Smith [23] as the cause of a brown rot of lemons in California. It has since been found in other countries on various kinds of citrus fruits, but oranges are more resistant to attack than lemons. The fungus affects the fruit, leaves, and twigs near the ground, the lower part of the trunk, and the roots. The first sign of attack is the browning of small groups of leaves on branches near the soil and the appearance of dead areas on the twigs, frequently accompanied by the exudation of gum. The bark of an infected trunk is killed down to the cambium, and gum exudes copiously from it, as described by Fawcett [24]. On the fruit the disease first appears as a brown, sunken spot, which spreads rapidly until the whole may be affected. Diseased fruits have a peculiar pungent odour. The disease may spread rapidly in storage if slightly affected fruits touch healthy ones.

The fungus is a saprophytic organism of the soil, thriving only under moist conditions which allow of the formation of zoospores. These are splashed by rain to the lower branches and fruits.

Owing to infection arising from the soil, the lower branches of the trees should be cut off up to a height of two feet. The splashing of zoospores upwards can largely be prevented in summer by constant cultivation of the surface soil, and in winter by the growth of a cover crop, e.g. *Melilotus indica* in California, and *Lotus hispidus* or rye grass in New Zealand. During storage, diseased fruit should not be allowed to come into contact with healthy fruit.

Phytophthora, de Bary

Conidiophores sympodially branched, occasionally unbranched ;
conidia * usually form zoospores on germination, but sometimes
germinate directly to form a mycelium ; oospores frequently occur,
the antheridia being either amphigynous or paragynous.

It is difficult to differentiate between the genera Phytopthora
and Pythium, but in the latter the antheridia are invariably para-
gynous.
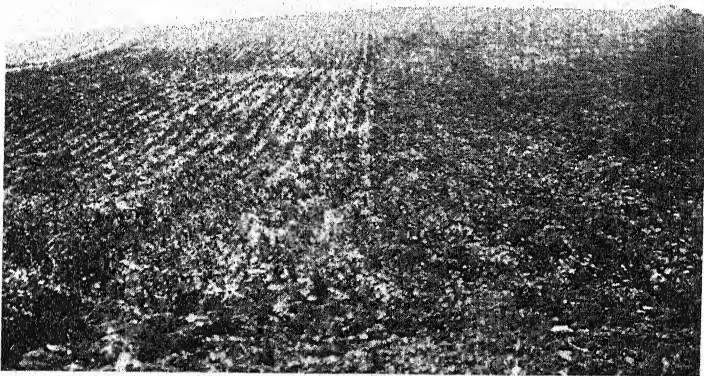
FIG. 10. Potato Blight, showing difference in attack on ' Abundance ' (left)
and ' Kerr's Pink ' (right).   (*A. Smith.*)

Phytophthora infestans, (Mont.) de Bary   Potato Blight.

Conidiophores single or in groups, sympodially branched, swollen
at intervals, with the conidia ultimately chiefly lateral ; conidia
inverted pear-shaped, pointed at one end, 27–30×15–$20 \, \mu$, forming
6–16 zoospores on germination. Oospores occur in cultures, but
have only recently been found in nature [26a].

Potato Blight was first recorded in Europe about 1840, after

* These bodies are called conidia by the writer, because they are dissemi-
nated as single units, although they generally behave as sporangia on
germination.

which it developed rapidly, causing destructive epidemics in 1845–6 and at later periods. It is now widespread wherever potatoes are grown, but varies greatly in intensity from season to season and place to place, according to weather conditions.

Fig. 12. Conidiophore of *Phytophthora infestans.* × 100. (*R. W. Marsh.*)

Fig. 11. Under-surface of potato leaflet affected by Blight. Natural size. (*G. H. Pethybridge.*)

The first sign of attack is the appearance of brownish-black spots on the leaves and stems, which may spread rapidly until the whole of the haulm is blackened. From the under surface of the spots conidiophores arise as a whitish mould under humid conditions. The conidia, blown about by wind, infect other leaves by liberating zoospores under moist conditions. The zoospores, on coming to rest in the film of dew or rain-water on the leaf surface, put forth germ-tubes which penetrate the epidermis directly or pass through the stomata, thereby establishing a mycelium in the tissues, which quickly die.

The tubers become infected by zoospores liberated from conidia which have fallen from the haulm to the soil, through

which they are washed down to the tubers by rain. The tubers can be infected either through the lenticels or eyes, or through wounds. The mycelium in the tuber is chiefly intercellular, and the death of the invaded tissue causes a brown discoloration which can be seen through the skin. The rot of potato tubers caused by *P. infestans* is of the nature of a true dry rot, but when infected tubers remain under specially moist conditions, secondary organisms, particularly saprophytic bacteria, may lead to the development of a wet rot. Blight may develop in storage owing to the tubers being exposed to contamination by conidia or zoospores during lifting, but it does not spread from tuber to tuber in storage.

Potato Blight is most likely to occur epidemically during wet or muggy weather in the summer, provided the temperature is not too low. Except with the few very resistant varieties there is no exact correlation between the intensity of attack in the haulm and that in the tubers, although attacks of Blight late in the season are generally most dangerous for tuber infection. On clay soils the tubers are often more badly affected than on sandy soils. Löhnis[25] attributes this to the slighter suberization of the lenticels in clay than in sandy soils.

The varieties 'Champion II', 'President' ('Paul Krüger'), and 'Evergood' are markedly resistant to blight, but most other varieties commonly grown in Britain are more or less readily susceptible. In Holland the variety 'Bravo' is very resistant to attack, and Löhnis[25] has suggested that, as regards its tubers, this is partly due to some protoplasmic property of the cork cambium.

The mode of over-wintering of this fungus is still somewhat obscure. Clinton[26] and others have obtained oospores in cultures, and Murphy[26a] has also found these on rotten tubers, but the germination of these resting bodies has not yet been seen. Field observations, such as those of Brooks[27], appear to indicate that some form of the fungus may persist in the soil in certain areas from season to season. de Bruyn[28] has cultivated the fungus on sterilized plant tissues and on sterilized soil, but it is not yet known how long the mycelium continues

to live in soil containing other organisms. She found that in these pure cultures the fungus, either in the form of oospores or other thick-walled resting cells, endured temperatures of $-20°$ to $-26°$ C. Melhus [29], Pethybridge [30], and others have shown that a small percentage of slightly blighted tubers produce aerial shoots bearing conidiophores, which may be the source of an epidemic. Thus mycelium perennating in the tubers is certainly one of the sources of infection year after year.

Early varieties of potatoes usually escape attack because they are lifted before Blight becomes epidemic. Deep earthing-up tends to prevent infection of the tubers. In wet, low-lying districts, where Blight recurs yearly, the haulm should be sprayed with one per cent. Bordeaux * or Burgundy mixture before Blight appears, a second spraying being given about three weeks after the first application. In this way the haulm is protected, and the danger of tuber infection is minimized. An increased yield of $1\frac{1}{2}$ to 2 tons of sound tubers per acre is frequently obtained in sprayed crops. In England it is particularly in the west and south that protection against Blight by spraying is advisable. In districts where the recurrence of Blight in epidemic form is less regular, it has not yet been definitely ascertained by experiment whether, over a period of years, it pays to spray the crop. Potato haulm badly affected with aphides and crops in the vicinity of smoky towns should not be sprayed, as under these conditions the fungicide scorches the foliage.

The crop should be lifted and stored when there is no abundant supply of conidia in the air or in the soil, otherwise the tubers, although apparently sound, may in reality be slightly infected and may develop Blight in storage, as pointed out by Murphy and McKay [31].

Phytophthora infestans also occurs on the shoots and fruit of tomato plants late in the season, but this form of the fungus probably differs biologically from that on the potato, as indicated by Berg [31a] and others.

* See p. 362.

Phytophthora Phaseoli, Thaxter

Conidiophores simple or once dichotomously branched, and once to several times successively inflated below their apices ; conidia with truncate base and papillate apex, $35-50 \times 20-24 \mu$.

Leonian [22] considers this fungus to be a variety of *P. infestans*.

Thaxter [32] has investigated this fungus, which attacks the pods and leaves of Lima beans in the United States. Sturgis [33] states that the disease is commonly spread by bees carrying spores to the young ovaries in the course of their visits to flowers.

Phytophthora erythroseptica, Pethybridge   Pink Rot of Potatoes.

Conidia bluntly pointed at one end or nearly ovoid, $30 \times 20 \mu$; oospores spherical, $29-30 \mu$.

Pethybridge [34] has described this fungus, which is the cause of a rot of potato tubers in Ireland, where greater losses are sometimes due to it than to *P. infestans*. The disease also occurs rarely in other parts of the British Isles, Holland, Switzerland, and Java. The same fungus, or a closely related form, causes a wilt of *Atropa belladonna*.

Infection takes place from the soil upon germination of the oospores. Tubers invaded by the mycelium, which enters by way of the stolons, become affected by a firmish soft rot, the tissues becoming pink on exposure to the air, and finally black. The fungus attacks other underground parts of the plant, and the whole plant may wilt if the stems are affected. Conidia are rarely formed except when a copious supply of water surrounds the diseased parts, but oospores occur in the tissues and subsequently pass into the soil.

The disease is serious only in wet districts where potatoes are grown on the same land year after year. The oospores may retain their vitality for at least four years, hence the rotation should be longer than this. Diseased plants and tubers should be burnt to prevent the oospores from contaminating the soil. No varieties of potatoes are known which are immune from the disease.

Phytophthora parasitica, Dastur (= *P. terrestris*, Sherbakoff)

Conidiophores generally unbranched, bearing a single terminal roundish or ovoid conidium, which may form zoospores or germinate directly; conidia $25–50 \times 20–40 \mu$, papillate, stalkless; chlamydospores yellowish, thick-walled, spherical, $20–60 \mu$; oospores known only in cultures, spherical, $12–35 \mu$.

In cultures on standard oat agar there is abundant white, lanate, aerial mycelium, which produces conidia sparingly.

Dastur [25] states that this fungus kills castor-oil seedlings and attacks the leaves of older plants in India, where great damage is done by it, especially in damp, low-lying situations. According to Ashby [26], it induces a leaf-stalk disease of the coco-nut palm in Jamaica. Stevens [37] states that it causes 'foot-rot' or 'mal di gomma' of citrus trees. This disease of citrus trees is characterized by the drying-up or rotting of the bark of the collar and crown roots, which is accompanied by the exudation of gum. Infection occurs from the soil, and heavy soils and imperfect drainage favour the disease. The sour orange is very resistant to attack, and susceptible kinds of citrus (e. g. sweet orange) should be 'worked' on it.

In the cultivation of tomatoes under glass, *P. parasitica* is one of the causes of 'damping-off' and 'foot-rot', which can usually be controlled by watering the soil with Cheshunt compound (see p. 366). Sherbakoff [28] states that this fungus is also responsible for the 'buck-eye' rot of fruits near the ground, which commences as a brown discoloration at the stylar end just before ripening. In tomato cultivation under glass this disease of the bottom trusses of fruit may arise by the splashing upwards of spores from the soil through careless watering.

Ashby * considers that *P. Nicotianae*, which causes a serious 'damping-off' disease in tobacco nursery beds and a blackening of the shoots of adult plants, is identical with *P. parasitica*.

Phytophthora cryptogea, Pethybridge and Lafferty

Conidiophore branches long and straggling; conidia inversely pear-shaped, $24–50 \times 17–30 \mu$, apex obtuse, non-papillate, forming zoospores on germination; oospores yellowish, spherical, 25μ.

* *Trans. British Mycological Society*, vol. 13, p. 86, 1928.

Pethybridge and Lafferty [39] have described this species as the cause of 'foot-rot' of young tomato plants grown under glass. A similar disease of young plants of potato, petunia, China aster (in which the disease is called 'black-neck'), and wallflower is also caused by it. In addition, the organism is one of the fungi responsible for the 'damping-off' of tomato seedlings; it also causes 'shanking' of tulips.

Infection occurs at about soil-level from spores in the soil, and a rot of the roots and the lower part of the stem is ultimately induced, which causes the plant to collapse. The disease sometimes occurs epidemically in intensive tomato cultivation. Under glass the disease can be prevented by sterilizing the soil with steam or by watering it with Cheshunt compound * during the growth of the young plants. It is important that the water-supply should not be contaminated with the spores of the fungus. The optimum temperature for the growth of this fungus is about 25° C. (77° F.); if the disease develops, the temperature should be kept as low as possible without injuring the crop. Young plants, not too badly affected, may be cut off above the decayed part and treated as cuttings, when they will often put forth new roots and grow into healthy plants.

Phytophthora Faberi, Maublanc

Conidiophores terminated by a single conidium, more rarely branching and bearing two conidia, devoid of swellings below the conidia; conidia generally lemon-shaped with an apical papilla and a short pedicel, $30–80 \times 25–42\,\mu$, average $48 \times 28\,\mu$; chlamydospores yellowish when old, spherical, $22–50\,\mu$, average $38\,\mu$; oospores spherical, $21–28\,\mu$, formed only when two strains are mixed together.

In cultures on standard oat agar conidia are formed profusely.
Ashby † considers this species to be identical with *P. palmivora*.

This fungus causes serious diseases of rubber (*Hevea brasiliensis*), cocoa, coco-nuts and other tropical plants. It has been closely studied by Gadd [40]. On rubber Petch [41] states that it

* See p. 366.
† Communication made to the British Mycological Society, November 1927.

kills the bark, the affected parts of untapped bark being claret-coloured just under the surface. Such bark does not yield latex and should be excised, the exposed tissues being painted with an antiseptic. The fungus also attacks the fruits, inducing a rot. The same species, but probably a different biologic form, causes a disease of the bark of cocoa trees ('canker') and a brown rot of the pods, especially in overcrowded plantations. In well-managed cocoa plantations the disease can be controlled, according to Nowell[42], by spraying with Bordeaux mixture. Reinking[43] states that *P. Faberi* is one of the causes of bud-rot of coco-nut palms.

Phytophthora Meadii, McRae

Conidia usually single on the conidiophores, $35 \times 25 \mu$, forming zoospores on germination; chlamydospores roundish, $17–34 \mu$; oospores spherical, $16–32 \mu$, average $20·75 \mu$.

This fungus, which is closely related to the previous species, attacks Hevea rubber in Southern India and Burmah. It has been described by McRae[44]. It causes the 'black thread' disease of newly tapped bark, which is characterized by the presence of vertical, black lines, parallel to one another, just above the tapping cut. It also causes a rot of the fruits, leaf-fall, and a die-back of the twigs. Leaf-fall, induced by this fungus, greatly reduces the output of latex; in Southern India attemps are being made to prevent this leaf-fall by spraying with Bordeaux mixture. The 'black thread' disease of the bark can generally be controlled by painting the tapping cut after each tapping with an antiseptic, e.g. brunolinum, carbo-lineum, or solignum, but during the height of the wet season in Burmah it is advisable to stop tapping.

Phytophthora palmivora, Butler

This fungus was first described as *Pythium palmivorum*, but Butler has transferred it to the genus Phytophthora.

Conidia formed in the weft of mycelium and not on special conidiophores, inverted pear-shaped, papillate with a short pedicel, $38–72 \times 33–42 \mu$; oospores spherical, $35–45 \mu$.

This species is closely related to the two previous forms,

and it is probably identical with *P. Faberi*. Butler [45] has shown that it causes 'bud-rot' of Palmyra palms in India. It may also be one of the causes of 'bud-rot' of coco-nut palms, as indicated by Gadd [46]. The tissues attacked by Phytophthora are invaded by secondary organisms, causing the bud to become putrid.

'Bud-rot' of coco-nut and other palms is of common occurrence throughout the tropics. As reported in a discussion on the subject by Butler and others [47], the symptoms of 'bud-rot' are probably induced by several different causes, including growth in unsuitable soils and attack lower down the stem by another disease, as well as by direct parasitic invasion of the bud. Gadd [46] points out that in true 'bud-rot' the bud becomes rotten through a primary infection in or near the bud, before the older leaves are affected. He suggests that the term 'wilt' should be applied to other diseases of palms in which rotting of the terminal bud is preceded by the drooping and wilting of the older leaves.

Phytophthora Fagi, Hartig

A disease of beech and other seedlings is caused by this fungus, particularly in forest nurseries, as described by Hartig [48]. Infection occurs through the germination of oospores in the soil. 'Damping off' may result, or the cotyledons and first-formed leaves may be killed or spotted. The disease is most severe in wet and shady situations. It is sometimes advisable to partly sterilize the seed bed with weak formalin or sulphuric acid (see p. 367).

Phytophthora Cactorum, (Leb. & Cohn) Schroet.

Conidiophores rarely branched, conidia ellipsoidal or oval, 50–90 × 35–40 μ, with a prominent apical papilla; oospores spherical, 24–30 μ.

This species is closely related to the previous one. It causes a disease of cactus seedlings and has also been reported by Wormald [49] as producing a rot of apples and pears in England.

Phytophthora Syringae, Klebahn

Conidiophores formed sympodially, bearing 1–7 conidia; conidia 40–75 × 30–42 μ; oospores spherical or seldom oval, yellowish, smooth, 18–36 × 17–25 μ.

Klebahn [50] has investigated a disease of lilac caused by this species. The disease is most common on lilacs which are 'forced' in order to form early blooms. The buds and tips of the shoots may be killed, and large diseased patches may arise on the older stems. Oospores are formed in the tissues. Klebahn [50] found that where lilacs were 'forced' on a large scale the plants were often laid on soil contaminated with oospores of the fungus, from which infection arose.

Lafferty and Pethybridge [51] have proved that this species causes a rot of apples in Ireland.

Phytophthora hibernalis, Carne

Conidiophores simple, bearing a single terminal conidium ; conidia elliptical, papillate, $17–56 \times 10–28 \mu$, deciduous, with persistent pedicels $2–5 \mu$ long. Antheridia amphigynous, rarely paragynous ; oospores spherical, $22–45 \mu$, yellow to tawny when mature.

Until recently a disease of citrus shoots and fruits in Australia has been confused with that caused by *Pythiacystis citrophthora*. Carne [52], however, has shown that the Australian disease is caused by a distinct fungus, which he names *Phytophthora hibernalis*. The same species has also been found on citrus trees in the Mediterranean region and in the Channel Islands.

In Western Australia the fungus is of serious importance. It attacks the leaves more frequently than the fruit, sometimes causing complete defoliation, and it is commoner on oranges than on lemons. The lower leaves are first attacked. Small twigs and branches may also be killed. Affected oranges show a dull brown area on one side, which gradually spreads over the whole fruit. Ultimately the fruit shrinks to a hard mummy, although the symptoms are often complicated by secondary organisms. Unlike the brown rot of citrus fruits caused by *Pythiacystis citrophthora*, this disease does not readily spread in storage.

The disease is active only in the cooler months, under conditions of high atmospheric and soil humidity. The disease is more prevalent on flat coastal lands than on elevated ground inland. It can be controlled effectively by spraying with

Bordeaux or Burgundy mixture before the cool wet season begins.

Phytophthora Richardiae, Buisman   Root rot of Arum.

Buisman [52a] has investigated a serious rot of the roots and corms of Richardias (Callas) in Holland, which he attributes to this species of Phytophthora.   The first effect of the disease is to cause a yellowing of the foliage, which is followed by a withering of the leaf-stalks ; affected plants remain small and produce only poor blooms.   The disease is propagated by planting slightly affected corms.   Buisman recommends the following control measures : upon lifting, affected corms should be washed vigorously with water and the rotten parts scraped away ; the corms should then be immersed in formalin (1 part to 49 parts of water) for an hour, and planted again in sterilized soil.

Salmon and Ware [52b] have described the occurrence of this disease in England.

Sclerospora, Schroeter

Conidiophores monopodially branched, sometimes sparingly produced ; conidia germinate to form zoospores, or, in some species, hyphae ; oospore wall united with that of the oogonium.   .

Sclerospora graminicola, (Sacc.) Schroeter

Conidia 19–31×12–21μ ; oospores pale brown, spherical, 23–25μ.

This fungus attacks various kinds of millets (including sorghum, bajra, and Italian millet) and is of most importance in India.   Diseased inflorescences may be sterile, and in bajra they are often hypertrophied, the ovaries being transformed into green shoots.   The leaves also are attacked ; these are marked by broad, white streaks, which change later to brown, and may become disintegrated.   A delicate downy growth of conidiophores appears on the affected parts, and oospores are produced in enormous numbers in the tissues.   The fungus probably survives from season to season by means of oospores. Biologic forms exist, as the fungus cannot pass from bajra to sorghum.   The disease is most prevalent on badly drained land.   The same species has recently been found on maize in the United States by Melhus and van Halten [53], where it stunts or kills young plants.

Sclerospora macrospora, Sacc., attacks wheat, oats, and maize in Italy, wheat and maize in the United States, wheat in Australia, and rice in Japan. It occurs also on wild grasses. Oospores only are known. The parts of wheat plants affected by this fungus are abnormally stiff and fleshy; the ears may be longer than usual, or branched and twisted, bearing leaf-like outgrowths, or the spikelets may be rudimentary. Noble[53a] states that in Australia the disease occurs only under excessively wet conditions.

Sclerospora maydis, (Rac.) Butler

Butler[54] has described the effect of this fungus on maize in India; it checks the growth of the plant and causes the formation of pale streaks on the upper leaves. Affected plants usually produce no grain. Conidia only have yet been observed.

Sclerospora philippinensis, Weston, occurs commonly on maize in the Philippines, as reported by Weston[55]. Infected plants show a yellowing of the leaves in stripes and abortive development of the cobs, resulting in partial or complete sterility. The conidia, which so far have alone been seen, germinate by the formation of hyphae.

Plasmopara, Schröter

Conidiophores monopodially and freely branched, the extremities being obtuse; conidia form zoospores on germination; oospores free from the oogonial wall.

Plasmopara viticola, Berl. and de Toni.   Downy Mildew of the Vine.

Conidiophores fasciculate, 4–5 times branched, conidia ovate-elliptic, $12–30 \times 9–12 \mu$, germinating to form zoospores; oospores brown, spherical, $30–35 \mu$.

The downy mildew of the vine was introduced into Europe from the United States about 1878. It spread rapidly through all vine-growing countries in Europe, causing severe epidemics, and it occurs now in practically all vine-growing countries. It has only occasionally been recorded in England, as pointed out by Harrison and Ware[55a]. The fungus is found on both wild and cultivated grapes and on species of Ampelopsis. It affects the young stems, leaves, and fruits, penetrating all the tissues except the xylem, and sometimes living perennially in the twigs, according to Istvanffi[56]. The haustoria

are small and spherical. New infections occur by zoospores formed from conidia, the germ-tubes penetrating chiefly the lower epidermis by way of the stomata. The first sign of attack is the appearance of yellowish or brownish, angular spots on the lower leaves, from the under side of which a delicate white down develops, consisting of conidiophores protruding through the stomata. The spots may spread until the whole leaf is involved, when it falls prematurely. The disease develops rapidly in wet weather. Oospores may be formed in the tissues in the autumn and fall with the leaves or diseased grapes to the ground. The oospores germinate in spring by means of a short germ-tube, which immediately forms a conidium. Zoospores formed in such conidia are splashed by rain on to the lower leaves, and in this way the first infections are usually brought about. A severe leaf attack greatly weakens the vines. The fungus affects the fruit when young, preventing its proper development and rendering it worthless.

The disease varies in intensity from season to season, according to weather conditions, wet weather greatly favouring an epidemic. In connexion with this disease the fungicidal value of copper compounds was accidentally discovered in France, from which, chiefly through the work of Millardet, Bordeaux mixture was developed as a protective spray in 1881. Since then, spraying with copper fungicides has become part of the routine of viticulture in Europe. By careful consideration of meteorological conditions in spring and early summer it is possible in some countries (e. g. Hungary) to predict the date at which spraying of the vines should commence. To be effective, the fungicide must be applied before the spores are abundant in the air.

Pseudoperonospora, Rostowzew

This genus agrees with Plasmopara except that the conidiophores branch dichotomously. The genus is synonymous with Peronoplasmopara, (Berl.) Clinton.

Pseudoperonospora cubensis, (B. and C.) Rostowzew   Downy Mildew of Cucumbers.

Conidiophores grey to pale violet in mass, conidia ovoid to ellipsoid,

$20–40 \times 14–25\ \mu$; oospores spherical, yellowish, $30–43\ \mu$, maturing in decaying leaves.

This disease attacks cucumbers and melons, particularly under glass. It commonly occurs in the United States, as described by Clinton [57], and has been recorded in Japan, but it has not yet been reported in England. When the foliage is badly attacked few cucumbers are formed, and these are so misshapen as to be unsaleable. The disease can be controlled by spraying with Bordeaux mixture.

Pseudoperonospora Humuli, (Miyabe and Tak.) Wilson Downy Mildew of Hop.

Conidiophores violet-black in mass, protruding in groups of 2–5 from a stoma, $200–460\ \mu$ long, $6–7\ \mu$ wide, 5–6 times dichotomous, rather spreading, ultimate branches tapering to a blunt point; conidia broadly elliptical or obovate, of light smoky colour, $22–26 \times 15–18\ \mu$, with a blunt apical papilla; oospores spherical, smooth, light brown, $28–34\ \mu$ in diameter.

Although known for some years in Japan, where it was first described by Miyabe and Takahashi [58] in 1905, and in the United States (1909), this fungus was not found in England until 1920, where it has been studied by Salmon and Ware [59]. Ducomet [60] has recorded it in France, and it occurs also in Belgium, Germany, Russia, and other European countries. Ware [61] has shown that the fungus is perennial in the root-stock of wild and cultivated hops, from which stunted shoots or 'spiked growths' containing mycelium arise in the spring. The leaves of these 'spiked growths' are small and brittle; the upper surface appears silvery, while the under surface is violet-black with conidiophores. Later in the season the extremities or lateral shoots of bines, 5 to 7 feet high, may be similarly transformed, and their growth then ceases. If, however, these terminal 'spikes' be cut off, healthy laterals will arise lower down the bines, which can be trained upwards. Affected tissues are discoloured brown. The mycelium is found in the cortex, pith, and medullary rays of the stem, and may occur even in the roots. The haustoria are lobed. Oospores are formed in the pith, especially in the hollow parts, in the leaves, and in the 'cones'.

In a wet season the fungus occurs epidemically on the leaves and 'cones' through widespread distribution of the conidia. Numerous angular, brown spots appear on the leaves; the 'cones' change colour from light green to brown, and, if affected when young, they may fall prematurely or be deformed. Badly affected 'cones' are of little or no value for brewing. Salmon [61a] states that the 'cones' of the variety 'Fuggles' are practically immune from attack.

'Spiked growths' should be cut off in the spring and killed by immersion in a disinfectant. If only a few hop 'hills' in a garden are affected they should be destroyed, and wild hops should not be permitted to grow in the vicinity of hop gardens. In planting new 'hills' care should be taken to use only sets from healthy nurseries. Blattny [61b] states that on the Continent considerable success has been obtained in the control of this downy mildew by spraying repeatedly with Bordeaux mixture. Under English conditions Salmon [61a] points out that hops should not be sprayed when in flower, nor after the 'cones' have formed.

Peronospora, Corda

Conidiophores dichotomously branched, conidia germinating by means of a germ-tube; haustoria frequently branched.

Peronospora parasitica, (Pers.) Tul.

Ultimate branchlets of the conidiophores curved, conidia broadly elliptic or nearly globose, hyaline, $24-27 \times 12-22 \mu$; oospores yellow-brown, spherical, $26-45 \mu$.

This fungus is of cosmopolitan distribution on wild and cultivated members of the Cruciferae, including Brassicae and wallflowers. It may cause marked hypertrophy of the host, especially when the stem or inflorescence is attacked. It is occasionally of importance in the cultivation of cauliflowers and wallflowers. Gardner [62] states that the mycelium may over-winter in turnip roots.

Gäumann [63], who has recently monographed the genus, considers the forms on Brassicae and wallflowers to be specifically distinct from *P. parasitica* on *Capsella bursa-pastoris*. He names these forms *P. Brassicae* and *P. Cheiranthi*.

Peronospora Schleideni, Ung.   Downy Mildew of Onions.

Conidiophores faintly purple in mass, conidia obovate to pyriform, $45-58 \times 20-25 \mu$; oospores light brown, globose, 30μ.

The downy mildew of onions occurs commonly wherever onions are grown on a large scale; it is frequently epidemic in a wet summer. Affected leaves become pale and wilt, and bear a delicate purplish-grey bloom of conidiophores. Severe attack cripples the growth of the bulbs. Diseased foliage is often secondarily attacked by mould fungi, which cause a blackish discoloration. Oospores are formed in the tissues. The mycelium grows down into the bulbs, where it may persist over the winter, as pointed out by Murphy [64]; onions containing perennial mycelium, if used for seed purposes, produce conidiophores on the new leaves, and may be the means of starting an epidemic. Bulbs for seed should be saved only from healthy crops.
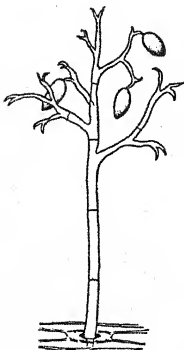
Fig. 13. Conidiophore of *Peronospora Schleideni* protruding from stoma. ×175. (*R. W. Marsh.*)

Peronospora Spinaciae, Laubert (= *P. effusa*, Grév. f. *Spinaciae*, Rab.)

The conidiophores are pale violet when seen in mass. The fungus may cause a serious disease of spinach. According to Gäumann [63] it is confined to the genus Spinacia, but very closely related forms occur on other members of the Chenopodiaceae.

Peronospora Schachtii, Fuckel   Downy Mildew of Beet and Mangold.

Conidiophores densely aggregated, 6-8 times dichotomized, conidia ovate, pale brown, $20-24 \times 15-18 \mu$.

This fungus attacks sugar-beet and mangolds on the Continent, and it has been recently recorded in England by Salmon and Ware [65] and others. It may harm the young leaves

considerably, which become yellowish-green, thickened, and curled, bearing on the under surface a buff-grey bloom of conidiophores. Oospores have been found on the Continent. The mycelium is sometimes perennial in the root-stock. Plants for seed purposes should be saved only from healthy seed beds, which should be placed as far as possible from field crops of mangolds and beets. If the disease appears in a crop grown for seed, the affected plants should be destroyed.

Peronospora sparsa, Berk. Downy or Black Mildew of Roses.
Conidiophores scattered, conidia broadly elliptic, $17-22 \times 14-17 \mu$; oospores brown with a stratified wall, $30-34 \mu$ in diameter.

Roses under glass are not infrequently affected by this fungus, which causes the formation of irregular, brown spots on the young leaves, from the under surface of which minute, scattered conidiophores arise. The spots on the leaves extend rapidly and the leaves often fall; young stems may also be affected. The disease can be controlled by spraying with Bordeaux mixture or by dusting with flowers of sulphur.

Peronospora Trifoliorum, de Bary, occurs on cultivated clovers, lucerne, soya beans, and wild leguminous plants. According to Gäumann [63] this is a complex of species, including Sydow's species *P. Trifolii arvensis, P. pratensis, P. aestivalis, P. manshurica,* &c.

Peronospora Viciae, (Berk.) de Bary   Downy Mildew of Peas and Vetches.

The foliage and pods of peas, especially mid-season varieties, are sometimes affected by this downy mildew, which appears as a greyish-brown growth. Diseased pods are often empty and coated inside with masses of oospores.

Gäumann [63] considers that *P. Viciae,* (Berk.) de Bary, is an aggregation of species, including *P. Pisi,* Sydow, *P. Viciae,* (Berk.) Gäumann, *P. Viciae sativae,* Gäum., *P. sepium,* Gäum., &c.

Bremia, Regel

Conidiophores with a pronounced sub-terminal swelling, from which arise a number of short branches, each bearing a conidium which germinates by means of a germ-tube.

Bremia Lactucae, Regel   Downy Mildew of Lettuce.

Conidia ovate, hyaline, 16–22×15–$20\,\mu$; oospores light brown, wrinkled, spherical, 26–$35\,\mu$.

Lettuce grown under glass is often affected by this fungus, which also occurs more rarely out of doors. Affected leaves become yellowish or sometimes brown, and may wilt. The disease can usually be controlled under glass by avoiding excessive humidity and high temperatures. In the Annual Report of the Cheshunt Research Station for 1926, Jagger states that varieties of lettuce resistant to *Bremia Lactucae* in California are susceptible to attack in England.

Trachysphaera, Tabor and Bunting

Conidiophores simple or branched, with a terminal swelling to which the conidia are attached by stalks; oogonia pyriform, tuberculate, containing one thin-walled oospore.

Trachysphaera fructigena, Tabor and Bunting

Haustoria absent; conidia hyaline, spherical, echinulate, 13–$48\,\mu$ (average $35\,\mu$); oogonia thick-walled, irregularly tuberculate, pyriform, $40 \times 24\,\mu$.

This fungus has been found by Tabor and Bunting[66] to be the cause of a disease of Liberian coffee and cocoa fruits on the Gold Coast. The fruits become discoloured and later are covered with a white or pink-brown incrustation of conidia. Wounded or moribund fruits are most liable to attack, and in wet seasons the whole coffee crop may be endangered. The mycelium is both inter- and intra-cellular. Oospores are formed in the tissues.

Cystopus, Persoon

Conidiophores unbranched, abstricting chains of conidia which produce zoospores on germination; oospores formed in the host tissues.

Cystopus candidus, (Pers.) de Bary   White Blister of Brassicae, &c.

Conidiophores hyaline, clavate; conidia spherical, 15–$18\,\mu$; oospores brownish, thick-walled, verrucose, 35–$40\,\mu$ in diameter.

This fungus attacks many wild and cultivated plants of the

family Cruciferae, including Brassicae, turnips, radishes, and mustard. It forms glistening white pustules on the leaves, stems, and inflorescences, sometimes causing hypertrophy. It
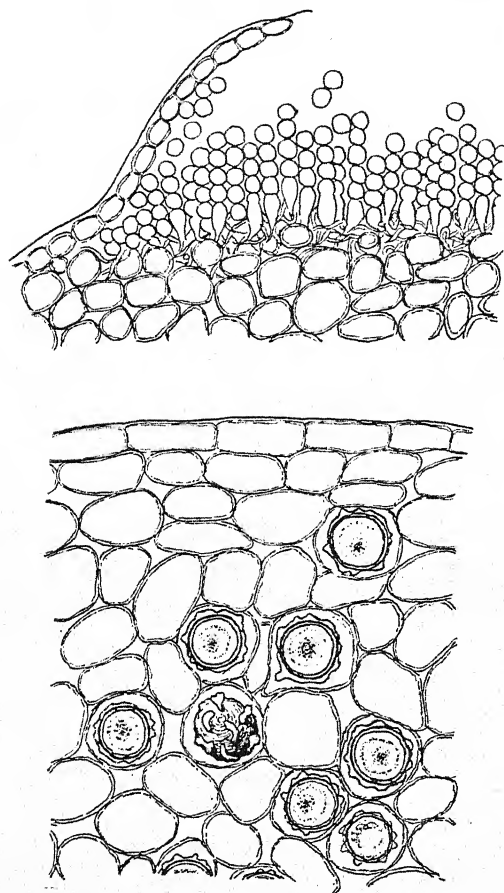
FIG. 14. *Cystopus candidus*, (above) section through a conidial pustule ; (below) section showing oospores in host cells × 175. (*R. W. Marsh.*)

is frequently accompanied by *Peronospora parasitica*. Eberhardt [67] and Melhus [68] have shown that biologic forms of the fungus exist. The fungus is of little economic importance, although it may cause Brassicae to look unsightly.

Cystopus cubicus, (Strauss) de Bary   White Blister of Salsify.

Conidiophores hyaline, conidia 18–22×12–$15\,\mu$; oospores dark brown, reticulate, spherical, 44–$68\,\mu$.

Salsify is the only food plant affected, but the fungus occurs on other members of the Compositae. The disease appears in the form of white spots on the shoots.

MUCORALES

Mycelium profuse; sporangia stalked, forming numerous non-motile spores; zygospores produced by the conjugation of two similar or nearly similar cells; many species exist in the form of two sexually differentiated strains, which generally differ only physiologically from one another (heterothallism).

Rhizopus, Ehrenberg

Aerial mycelium stoloniferous, sporangiophores arising from the nodes.

Rhizopus stolonifer, (Ehrenb.) Lind. ($=$ *R. nigricans,* Ehrenb.)

Sporangia spherical, black, columella hemispherical; spores hyaline, sub-globose, or irregular, 11–$14\,\mu$; zygospores black, warted, spherical, 150–$200\,\mu$.

Under certain conditions this mould causes a soft rot of tomato, apple, pear, and strawberry fruits, and of sweet potato tubers. It can obtain entry into the tissues only through wounds. Upon the mycelium passing into the tissues it secretes an enzyme which causes the dissolution of the middle lamella and consequently disintegration of the tissues. Mature fruit is much more liable to attack than immature fruit. If care is taken in picking and packing, this kind of rot can be reduced to negligible proportions. Sweet potato tubers should be lifted carefully and stored at as low a temperature as possible. Harter [69] states that other species of Rhizopus, e.g. *R. Tritici,* may also rot sweet potatoes in storage, entry being obtained through wounds.

Rhizopus necans, Mass.

This fungus is similar to *R. nigricans,* but the zygospores are covered with spinous warts.

According to Massee [70] this species causes a rot of the bulbs of *Lilium speciosum* and *L. auratum* in transit.

Mucor, Linn.

As in Rhizopus, but the mycelium is not stoloniferous.

Mucor spp.

Several species of Mucor cause rots of various kinds of ripe fruits, the fungus obtaining entry through wounds.

Choanephora, Cunningham

Sporangia of two kinds; large sporangia spherical, columella small, spores few; small sporangia clavate, one spored, borne in heads in the enlarged apices of umbellately branched sporangio-phores.

Choanephora infundibulifera, (Curry) Sacc., attacks the blossoms of Hibiscus in tropical Asia.

Choanephora cucurbitarum, (B. & Br.) Thaxter, causes a rot of pumpkins and other cucurbits in the United States.

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FUNGUS DISEASES (*continued*) : ERYSIPHALES, EXOASCALES, PLECTASCALES

ERYSIPHALES

MYCELIUM white or becoming dark coloured ; perithecia spherical, ovoid or flattened, usually without an ostiole, the asci being arranged in a definite layer at the base.

The most important family is the Erysiphaceae which comprises the true mildews (i. e. ' powdery ' mildews). These are nearly all white, surface parasites, with haustoria in the epidermal cells of the host. The conidiophores throughout this family are remarkably constant, being unbranched and abstricting hyaline, oval conidia in chains. The name Oidium was formerly used generically for the conidial stage. The members of the Erysiphaceae show a high degree of specialization in their parasitism, as demonstrated by Neger[1] and Salmon[2]. The family has been monographed by Salmon[3] and Jaczewski.*

Podosphaera, Kunze

Perithecia spherical or sub-spherical, containing one 8-spored ascus; appendages basal and apical, the latter usually dichotomously branched at the apex.

Podosphaera leucotricha, (Ell. & Ev.) Salm. Apple Mildew.

Conidia ellipsoid, truncate, $28–30 \times 12\,\mu$; perithecia $75–96\,\mu$, apical appendages often unbranched, upper part of perithecium becoming depressed at maturity ; ascus $50–70 \times 44–50\,\mu$, spores 8, $22–26 \times 12–14\,\mu$.

A serious disease of young apple shoots is caused by this fungus; pear and quince shoots are also occasionally affected. Buds on expanding in spring give rise to white mildewed shoots, which may be killed when severely attacked. Mildewed leaves often turn brown early in the summer. Fruit buds

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* Jaczewski, A. A., *Pocket key for the determination of fungi. Part II, Powdery mildew fungi* (in Russian). Leningrad, 1927.

may also be affected, and the blossoms killed. Woodward [4] has shown that infection occurs during the formation of new buds by means of mycelium on the shoots arising from buds infected the previous year, or by conidia then present in the air. The mycelium remains dormant in the bud until proliferation in the following year. During the summer the disease may spread to healthy shoots. Perithecia are formed both on the twigs and on the fruits, but under English conditions these are frequently sterile and the ascospores appear not to survive the winter. In other countries the ascospores may play an important part in causing infection. Most varieties of apples commonly grown in England are susceptible, but 'Worcester Pearmain' is somewhat markedly resistant. Woodward has proved that spores from apple can infect pear and quince.

In consequence of the manner of bud infection this is a difficult disease to control. The time from the appearance of mildewed shoots to the beginning of bud formation is only a week or two. Unless the buds can be protected from infection during the initial stages of formation, there is no further opportunity of control by fungicides, as the mycelium in the buds cannot be reached. Further investigation of this matter is necessary. On small areas the mildewed shoots should be cut off as soon as they appear, but it is hardly feasible to do this on a large scale except with bush trees.

Podosphaera Oxyacanthae, (DC.) de Bary   Hawthorn Mildew.

This is closely related to the previous species, but the equatorial appendages of the perithecia are 2–4 times dichotomously branched, the ultimate branches being more or less knob-shaped. The life-history is essentially the same as that of *P. leucotricha.* The hawthorn is commonly affected in England, but it is doubtful whether the perithecia remain viable over the winter. *P. oxyacanthae,* var. *tridactyla,* Salmon, affects young plum shoots, but is rarely seen except in nurseries and under glass.

Sphaerotheca, Lév.

Perithecia containing a single 8-spored ascus ; appendages floccose, basal, often interwoven with the mycelium.

Sphaerotheca Humuli, (DC.) Burr. Hop Mould or Mildew.

Perithecia 58–120 μ in diameter, appendages long, straight, septate, dark brown ; ascus broadly elliptic to sub-globose, 45–90 × 50–72 μ, spores 8, 20–25 × 12–18 μ.

Hop mould or mildew is perhaps the most serious fungoid disease of hops. It affects chiefly the leaves and young 'cones', rendering the latter useless; the intensity of attack varies greatly from season to season. Perithecia formed on the affected parts fall to the ground, where they remain dormant during the winter; those on the surface of the soil or remaining on the dead hop bines of the previous year eject the ascospores violently into the air in the following spring, and these bring about infection of the leaves. Abundant formation of conidia coupled with wet weather may lead to a serious epidemic. Hop mould can be prevented from seriously injuring the cones by dusting the bines with finely-divided sulphur as soon as there is the slightest sign of mildew on the leaves. Several dustings may be necessary, but the crop is so valuable that the grower can afford them. Lime-sulphur is not so effective a fungicide in this instance as sulphur. Wild hops should not be allowed to grow in the vicinity of hop-gardens.

Work is in progress under Salmon [5] to try to obtain commercial varieties of hops which will be more resistant to the fungus than those grown at present in England.

A biologic form of this fungus occurs on cultivated strawberries. Affected leaves tend to roll upwards from the margin, but the most serious injury is to the young fruit, which may be rendered unsaleable. The conidial stage on this host and the raspberry is inconspicuous. This mildew can be prevented from attacking the fruit seriously by dusting the plants once or twice early in May with finely divided sulphur.

In the United States a form of *Sphaerotheca Humuli* often occurs on roses.

Sphaerotheca mors-uvae, (Schw.) Berk. American Gooseberry Mildew.

Mycelium white, becoming brown, persistent; perithecia gregarious, more or less immersed in the mycelium, sub-globose, 76–110 μ ;

appendages few or obsolete; ascus $70–92 \times 50–62 \mu$, spores 8, 20–$25 \times 12–15 \mu$.

This fungus, which is indigenous on the gooseberry and other kinds of Ribes in the United States, was introduced into Europe (Northern Ireland) about 1900. It spread rapidly in England and other countries where gooseberries are grown on a large scale. This mildew chiefly affects the young stems

Fig. 15. *Sphaerotheca mors-uvae*, (right) perithecium with appendages, × 150; (left) ascus, × 300. (*R. W. Marsh.*)

and berries, covering them eventually with a brown scurf; it is somewhat infrequently seen on the leaves. Perithecia fall to the ground with the diseased berries, and from the autumn onwards they fall also from the persistent mycelium on the twigs. Infection in the spring occurs mostly from ascospores liberated from perithecia on the surface of the soil. The disease can be controlled by spraying the bushes with lime-sulphur of specific gravity 1·01, or 1·005 on the more delicate varieties, from the spring onwards until the berries are nearly fully grown. The last spraying before picking should be done with ammonium polysulphide, in order to prevent spotting of the berries, as advised by Eyre, Salmon and Wormald [6,7]. Nattrass [8] has obtained satisfactory control by spraying twice early in the season with ammonium polysulphide, and by a single early spraying with Burgundy mixture. It is sometimes advisable to cut off and burn the affected twigs in the early autumn as a means of reducing the number of perithecia. The most susceptible varieties of gooseberries in England are 'Whinham's Industry', 'Keepsake', 'Lancashire Lad', and the most resistant are 'Lancer', 'Crown Bob', 'Whitesmith'. Excessive nitrogenous manuring tends to increase the severity of this mildew through promoting succulent growth, which

readily succumbs to attack. The disease varies greatly in intensity from season to season. The earlier in the spring ascospore infection takes place the more likely is a serious attack to develop.

Red, black, and white currants, and the 'Worcester berry' are occasionally affected in England, but the disease is not so severe on them as on gooseberries.

Sphaerotheca pannosa, (Wallr.) Lév.   Rose and Peach Mildew.

Mycelium persistent on the stem, becoming buff-coloured; perithecia embedded in the mycelium, 85–120 μ in diameter; appendages few or absent; ascus averaging 100×60–75 μ, spores 8, 20–27 $\times$ 12–15 μ.
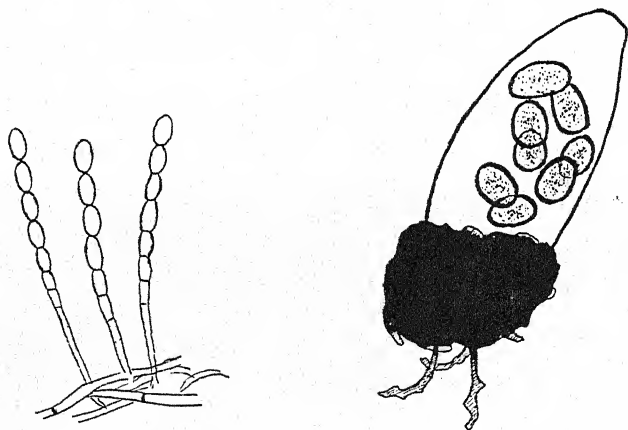
Fig. 16. *Sphaerotheca pannosa*, (left) conidiophores, $\times$ 375; (right) ascus protruding from perithecium, $\times$ 400. (*R. W. Marsh.*)

Mildew is one of the chief fungus enemies of the rose-grower. It occurs commonly on wild roses. Young stems, thorns, leaves, and fruits are affected. Roses which are badly attacked look unsightly and may lose their leaves prematurely, thereby weakening the growth. The fungus is spread rapidly during the summer by conidia. Perithecia are formed in the felted mycelium on the stems and thorns; these serve as a means of hibernation, the ascospores being liberated in the spring. In sheltered positions in England conidia may be formed through-

out the winter at the extremities of the shoots of susceptible varieties such as certain of the Ramblers. Rose mildew varies in intensity from summer to summer, and is often most severe in a dry season. There are great differences in varietal susceptibility, some of the Ramblers, e.g. Dorothy Perkins and Crimson Rambler, being among the most susceptible. Certain varieties are no longer grown because of their susceptibility to mildew.

The disease can be controlled by spraying, but repeated applications of the fungicide are necessary. Lime-sulphur of specific gravity 1·005 or weak sulphuric acid (1 part in 1,000 of water) can be used. A weak solution of carbolic acid (one dessert spoonful in a gallon of water), especially if two ozs. of soft soap per gallon is incorporated, also greatly checks this mildew. In greenhouses, mildew can be kept down by sulphuring or by painting the hot-water pipes with a mixture of sulphur and lime, the sulphur becoming gradually vapourized.

On peaches the fungus attacks chiefly the young stems and fruits. Varieties of peaches without glands at the base of the leaves are very susceptible to mildew, varieties with glands being highly resistant or immune.

The form of the fungus on the rose differs slightly from that on the peach, and infection from one host to the other does not occur. Rose mildew is sometimes distinguished under the name *S. pannosa*, var. *Rosae* Woonichine. In the United States *Sphaerotheca Humuli* also commonly occurs on roses.

Erysiphe, Hedwig

Perithecia containing several 2- to 8-spored asci; appendages floccose, similar to the mycelium and interwoven with it.

Erysiphe graminis, DC.   Mildew of Cereals and Grasses.

Mycelium more or less persistent, becoming brown; perithecia 135–280 μ in diameter; appendages rudimentary; asci 9–30, pedicellate, 70–108 × 25–40 μ; ascospores 8, rarely 4, 20–23 × 10–13 μ, seldom formed while the perithecia are attached to the host plant.

The haustoria are large, oval in shape, and provided with appendages, as described by Grant Smith [9].

The researches of Salmon [2] and Reed [10] have shown that this

mildew exists in a number of biologic forms, including those
on wheat, barley, oats, and rye respectively, to which each in
general is confined. Salmon [11] has pointed out, however, that
conidia from wheat can infect *Hordeum silvaticum*. He [12]
found also that by wounding barley leaves the fungus could
be transferred from wheat to barley and vice versa. 'Bridging
host' species may possibly exist, enabling the fungus to pass
from one group of hosts to another, as indicated by Salmon [2].
There are several biologic forms on wild grasses. Although
the fungus is strictly ectoparasitic in nature, Salmon [13] found
that it grew endophytically when spores were sown on the
injured mesophyll of appropriate leaves.
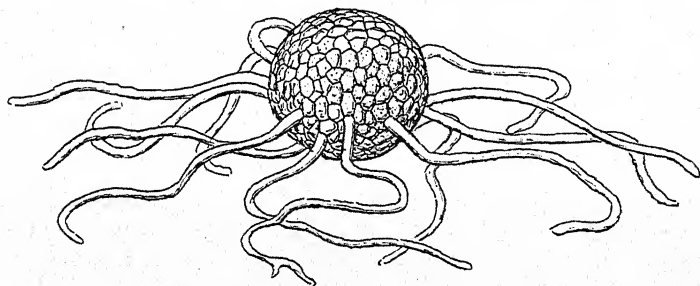
FIG. 17. *Erysiphe graminis*, perithecium with appendages. × 200.
(*R. W. Marsh.*)

Different varieties of cereals show marked differences in
susceptibility, 'Persian Black' wheat being immune. S. F.
Armstrong * has found that susceptibility to mildew in wheat
is a recessive Mendelian character. 'Norka' wheat sent to the
author by Professor E. B. Mains, of the United States, is very
resistant to mildew in England. Even under greenhouse con-
ditions the leaves are not affected, but mildew develops slightly
on the glumes as the ears mature. Biffen [13a] states that in a
cross between *Hordeum spontaneum* (resistant) and *H. hexasti-
cofurcatum* (very susceptible) the F_1 hybrids were very sus-
ceptible to mildew.

* Mr. S. F. Armstrong (National Institute of Agricultural Botany, Cam-
bridge) has kindly allowed me to use his unpublished results. He made
crosses between 'Persian Black' (immune) and 'Wilhelmina' and 'Rivet'
(susceptible).

The fungus affects the lower parts of the stems as well as the leaves. In some wheat varieties, e.g. ' Benefactor ', it occurs commonly on the glumes, where it is often followed by mould fungi such as *Cladosporium herbarum,* which cause blackening of the ears. Very susceptible varieties of cereals may be considerably weakened in their growth by mildew, and when the fungus attacks the stem severely it may be a factor in causing 'lodging' of the crop. Susceptibility is increased by heavy nitrogenous manuring.

On some English varieties of barley the mildew, instead of forming powdery patches, produces only ' sub-infections ', characterized by brown spots on the leaves with few conidiophores.

Under English conditions the conidial stage may be found on cereals and grasses in sheltered situations throughout the winter.

Erysiphe Polygoni, DC.   Mildew of Peas and Swedes.

A very variable species ; perithecia 65–100 μ in diameter ; appendages variable, more or less interwoven with the mycelium ; asci 2–8 or rarely up to 22, 46–72 × 30–45 μ ; ascospores 3–8, rarely 2, 19–25 × 9–14 μ.

This mildew occurs in a number of biologic forms on a wide range of hosts, the form on peas being unable to infect swedes and turnips.

In peas the fungus affects the pods as well as the leaves. It is particularly prevalent on late varieties, and may render them unprofitable. Perithecia are often found on this host.

The fungus commonly occurs upon swedes and turnips, especially in a dry summer, and the crops may appear white with it late in the season. This form of the fungus partially attacks Brassicae (e.g. kale), on which minute, discoloured sub-infections are formed, characterized by paucity of mycelium and few conidiophores. Perithecia are extremely rare in this form in England, and Searle [14] considers that the sub-infections on Brassicae, which survive the winter, may be a means of providing infectious material in the following summer.

Erysiphe Cichoracearum, DC.

Perithecia 80–140 μ in diameter ; asci about 10–15, more or less stalked, 58–90 × 30–35 μ, spores 2, rarely 3, 20–28 × 12–20 μ.

The hosts are numerous and diverse, including tobacco, cucurbits, and garden plants such as Phlox and Aster. Several biologic forms exist. It occurs commonly on cucurbits late in the season, but is rarely harmful. The Oidium stage of this mildew causes serious damage to tobacco in Nyassaland, Rhodesia, and Java.

Uncinula, Lév.

Perithecia with several 2- to 8-spored asci ; appendages simple or rarely dichotomously branched, uncinate at the apex.

Uncinula necator, (Schw.) Burr. Vine Mildew.

Perithecia 70–120 μ in diameter ; appendages variable in number and length, rarely branched ; asci 4–6, 50–60 × 30–40 μ, spores 4–7, 18–25 × 10–12 μ.
Conidiophores short, conidia 2- to 3-catenulate, 25–30 × 15–17 μ.

This mildew is of serious importance in viticulture through-out the world. It occurs also on wild species of Vitis and Ampelopsis. The conidial stage (formerly known as *Oidium Tuckeri*) is alone commonly found in Europe, where the disease was unknown before 1845. Perithecia were not found in Europe until long after its introduction. The fungus affects the leaves and flowers, causing a spotting of the former through the gradual death of the host cells. In the absence of perithecia the fungus may survive the winter by means of resistant cells found in the vegetative mycelium, as described by Appel [15].

This disease is best controlled by sulphuring (especially in greenhouses) or by the application of lime-sulphur of summer strength. Copper fungicides are not so effective in dealing with mildews of this kind as with 'downy' mildews. In greenhouses it is a good plan to paint the vine stems with a mixture of flowers of sulphur and soft soap during the dormant period ; when the temperature rises in the spring, the sulphur is gradually vaporized and effectively controls the mildew.

U. Aceris, (DC.) Sacc., occurs on sycamore and maples, the peri-thecia being formed chiefly on the under surface of the leaves.

U. Mori, Miyake, attacks mulberry leaves in Japan, occurring mostly on the upper side.

Microsphaera, Lév.

Perithecia with several 2- to 8-spored asci ; appendages repeatedly forked at the tip.

Microsphaera Grossulariae, (Wallr.) Lév. European Goose-berry Mildew.

Mycelium delicate, sometimes evanescent ; perithecia globose-depressed, 65–130 μ in diameter ; appendages 5–22, colourless, 4–5 times closely dichotomously branched ; asci 4–10, 46–62 × 28–38μ, spores 4–6, 20–28 × 12–16 μ.
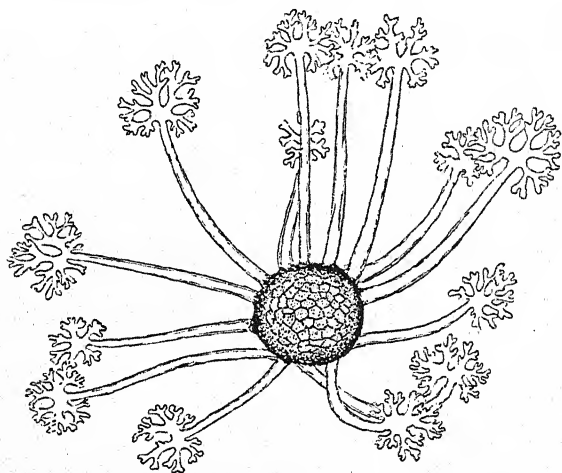
Fig. 18. *Microsphaera Grossulariae,* perithecium with appendages. × 150.
(*R. W. Marsh.*)

This fungus occurs commonly on gooseberry bushes late in the summer, particularly where they are in partial shade. It grows chiefly on the leaves, but is sometimes found on the fruit. This mildew is almost innocuous, but it may occasionally cause premature defoliation.

Microsphaera Alni extensa, Salm. (= *M. quercina,* (Schw.) Burr.) Oak Mildew.

Only the conidial stage of this fungus has yet been found in England, where it commonly occurs on the lower leaves of

oaks and on stool shoots. It has also been recorded on beech foliage. On the Continent perithecia have been found both on oak and beech. This mildew occurs commonly on oaks in the United States, but it was not seen in Europe until 1907. The fungus is rarely harmful, but where oaks are defoliated by caterpillars and the second foliage is attacked by mildew, Munro [16] states that the trees are greatly weakened and fall a prey to other fungi. The mode of overwintering of the fungus in England is unknown. Petri [17] has suggested that in Italy thick-walled cells (chlamydospores) in the mycelium may be a means of hibernation.

Phyllactinia, Lév.

Perithecia globose to lenticular, with many 2- or 3-spored asci; appendages equatorial, rigid, pointed with a bulbous base; apex of perithecium with a mass of densely-crowded, branched outgrowths.

The appendages show hygroscopic movements, which lift the perithecia from the leaf so that they fall to the ground.

Phyllactinia corylea, (Pers.) Karst.

Conidia solitary; perithecia scattered, $140-270\,\mu$ in diameter; appendages 5–18; asci 5 45, $60-105 \times 25-40\,\mu$, spores 2–3, 30–42 $\times 16-25\,\mu$.

Typical epidermal haustoria are not formed, but special hyphae pass through the stomata into the intercellular spaces, forming a limited internal mycelium with haustoria in the mesophyll cells, as described by Palla [18].

This mildew occurs frequently on the leaves of hazel, birch, ash, mulberry, and other trees, the perithecia being most abundant on the under surface. The mycelium is rather evanescent. The fungus sometimes causes defoliation.

Oidium, Link

This is a form genus including conidial stages of mildews, the perithecia of which have not yet been seen. There are many species, including several in the tropics, but most are unimportant.

Oidium Heveae, Arens

This occurs on the Para rubber tree in Java and Ceylon. When it attacks the young foliage appearing after 'wintering'

(= leaf-fall) of the trees, defoliation is again caused, which may adversely affect the latex-yield and growth. The mycelium is inconspicuous. Fortunately, this disease is not yet of widespread occurrence in rubber-producing countries. Until recently this mildew was unknown on *Hevea brasiliensis*, but forms of Oidium are of fairly frequent occurrence on other plants of the Euphorbiaceae in the tropics, and it is probably one of these which has begun to attack Hevea.

Oidium tingitaninum, Carter, occurs commonly on the leaves and young twigs of citrus trees in Ceylon, Java, and California.

Oidium Chrysanthemi, Rabenh., is sometimes abundant on chrysanthemums grown under glass, the lower leaves of which it may kill. This mildew can be controlled by sulphuring.

Oidium Euonymi-japonici, (Arc.) Sacc., is frequently seen in the south of England as a white, felted growth on the evergreen leaves of the Japanese Euonymus. The mycelium remains dormant in sheltered positions in England during the winter, and produces fresh conidia in the spring. This mildew occurs also on the Continent and in Japan.

The following genera belong to the family Perisporiaceae:

Zopfia, Rabenhorst

Perithecia black, with a few dark threads attached, opening irregularly at the apex; asci sac-like, large, 4- to 8-spored; ascospores with both ends sharply pointed, 2-celled, brownish-black.

Zopfia rhizophila, Rabenh.

This fungus was first found on dead asparagus roots in Germany, and it has recently been recorded on asparagus roots in England, where it may possibly be pathogenic.

Capnodium, Mont.

Mycelium black, effused; perithecia cylindrical, simple or branched, opening at the top by valves; asci elongated, spores 3–4 septate or muriform, brown. There are several conidial forms, which are often produced to the exclusion of perithecia.

Capnodium salicinum, Mont.

This 'sooty mould' grows in honey-dew deposited by aphides on the leaves of willow, poplar, and other trees. The mycelium is superficial and harmless.

Several other species of Capnodium occur on the leaves of trees, but they have not yet been fully investigated.

Diplocarpon, Wolf

Ascocarps with a distinct shield, which breaks away at maturity ; asci oblong to sub-clavate, 8-spored ; ascospores elongated, 2-celled, hyaline, paraphyses unbranched.

Conidia also occur.

Diplocarpon Rosae, Wolf   Black Spot of Roses.

Ascocarps epiphyllous, spherical to disciform, dark brown, 100–250 μ diam., opening stellately by rupture of the shield ; asci 70–80 $\times$ 15 μ ; ascospores oblong elliptical, unequally 2-celled, hyaline, 20–25 $\times$ 5–6 μ.

Conidial stage = *Actinonema Rosae,* Fr. Acervuli subcutaneous ; conidia 2-celled, often constricted, straight or subfalcate, hyaline, 18–25 $\times$ 5–6 μ.

' Black spot' of roses occurs wherever roses are cultivated. The disease is conspicuous on the leaves owing to the formation of purplish or purplish-black spots of irregular shape, the colour being due to the production of sap pigments in the host cells. Alcock [19] has recorded the disease also on young stems. In severe attacks the roses may be defoliated, but the fungus does little harm usually.   The young spots on the leaves are characterized by a fibrillose border, formed by radiating strands of mycelium below the cuticle.   Other hyphae penetrate the mesophyll. On older spots the acervuli appear as minute dark pustules which develop below the cuticle. Wolf [20] has found the perithecial stage on overwintered leaves in the United States.

When the disease is severe it can be considerably reduced by burning the affected leaves in the autumn, and by attention to pruning if the fungus is on the young stems. Massey [21] states that dusting with a mixture of nine parts of sulphur to one of lead arsenate during the summer controls the disease.

Meliola, Fr.

Mycelium effused, black ; perithecia globose, often with appendages; asci 2- to 8-spored ; ascospores oblong, 2- to 5-septate, rarely muriform, brown.

I

Meliola penzigi, Sacc.   Sooty Mould of Orange.

This fungus covers the leaves, young twigs and fruits of the orange and other kinds of citrus with a black film, growing in the honey-dew secreted by scale insects.   The only means of prevention is by the destruction of the insects, either by spraying with a miscible oil emulsion or by fumigation with hydrocyanic acid gas.   Attempts to parasitize scale insects artificially with fungi such as Aschersonia have not generally been successful on a large scale.

Webber [22] states that *Capnodium Citri* also commonly occurs as a sooty mould on citrus.

EXOASCALES

Mycelium parasitic in stems, fruits, and leaves, frequently causing hypertrophy ; asci formed in a palisade-like manner, either below the cuticle or under the epidermis, freely exposed when mature ; the ascospores often bud within the ascus.

Taphrina, Fries

Mycelium annual or perennial ; asci 4- to 8-spored, but the spores frequently bud within the ascus.

The genus Exoascus is sometimes separated from Taphrina, but on no valid grounds.

Taphrina deformans, (Berk.) Tul. (= *Exoascus deformans*, Fckl.)   Peach Leaf Curl.

Asci $25-40 \times 8-11 \mu$, usually containing 8 spores, which are subglobose, $3-4 \mu$ in diameter ; the spores occasionally bud within the ascus to form conidia.   In culture the conidia continue to bud in a yeast-like manner.

The fungus affects peach and nectarine leaves, causing the whole leaf or a part to become considerably thickened and curled ; it may also attack the flowers and young fruits.   The mycelium is sometimes perennial in the twigs, which may be killed by it.   The swollen leaves are often reddish in colour and become covered with a delicate bloom when the asci break through the cuticle.   After the ejection of the spores the diseased parts of the leaves become discolored owing to the
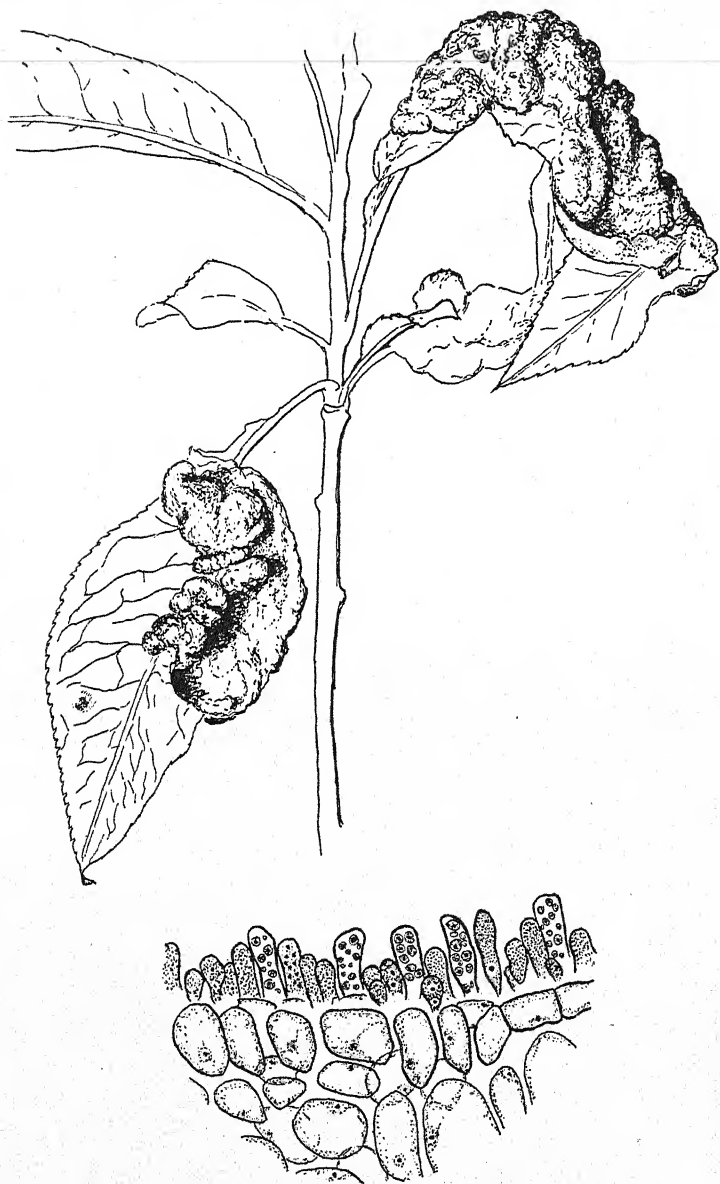
FIG. 19. *Taphrina deformans*, (above) on almond leaves, natural size; (below) section through affected peach leaf, showing asci. × 500. (*R. W. Marsh.*)

growth of moulds. As the disease can be effectively controlled by spraying the shoots, when the buds begin to swell, with Bordeaux or Burgundy mixture, or with lime-sulphur, infection must generally take place as the buds open. Direct infection of new foliage from perennial mycelium in the twigs rarely occurs. The exact mode of infection of the young leaves has not yet been fully elucidated, but presumably spores of the previous year, or hyphae formed from these, persist just within the buds or on them throughout the winter.

Wieben [23] succeeded in bringing about infection by inserting spores into young buds during the summer; in the following spring these buds gave rise to infected shoots.

The foliage of the almond tree is also commonly affected in England.

Taphrina Pruni, (Fckl.) Tul. (= *Exoascus Pruni,* Fckl.) Pocket Plums.

Asci elongate-cylindric, 30–60 × 8–15 μ, spores 8, globose 4–5 μ.

The disease known as Pocket Plums or Bladder Plums is common in some countries, but is rare in Britain except in the west and north. The fruits of *Prunus padus* and *P. spinosa* are greatly malformed by it. Less change in form is caused by the fungus in cultivated varieties of plum, but the stone does not develop. The fruit is permeated by the mycelium, and the asci, when mature, cover the fruit with a delicate bloom, which is soon replaced by moulds. The exact mode of infection is not yet known.

Taphrina Cerasi, (Fckl.) Sade. (= *Exoascus Cerasi,* Sade.) Witches' Broom of Cherry.

Mycelium perennial; asci clavate, 30–50 × 7–10 μ; the 8 ascospores bud in the ascus.

The presence of this fungus in the bark of wild and cultivated cherry trees causes the formation of densely crowded, erect branches in parts of the tree, known popularly as 'Witches' Brooms.' The leaves of the broom become invaded

by mycelium from the stems, and eventually a continuous layer of asci is formed on the under surface. The brooms should be cut out as they rarely fruit.

Taphrina minor, Sade. (= *Exoascus minor*, Sade.)   Cherry Leaf Curl.

This fungus also occurs on the cherry tree, but it does not form 'Witches' Brooms'. The leaves become blistered and curled ; the asci are formed on the under surface.

Taphrina insititiae, (Sade.) Johans., occasionally causes the formation of Witches' Brooms in domestic plums. In England it has been recorded only on the 'Damascene' plum in Worcestershire, where it is rather abundant.

Taphrina turgidus, (Fckl.) Sade. (= *Exoascus turgidus*, Sade.) Witches' Broom of Birch.

The densely crowded, affected branches of birch trees look like large birds' nests. The smaller twigs are rapidly killed by the fungus. The asci are formed on the leaves of living parts of the broom. In the birch, as well as in other trees, similar 'Witches' Brooms' are sometimes caused by mites.

Taphrina aurea, Sade.

This species forms large hypertrophied blotches on the leaves of the black poplar, which are golden-yellow on the spore-bearing surface when the asci are mature, in consequence of the presence of an oily pigment. The asci are chiefly produced on the under surface, and are full of minute spores when ripe. The upper surface of affected parts of the leaves is sometimes somewhat silvery in appearance. The disease varies greatly in incidence from season to season. Wieben [23] has shown that the fungus persists in the outer parts of the buds during the winter.

Taphrina bullata, (Berk.) Tul., occurs occasionally on pear and quince leaves, forming brownish blisters.

PLECTASCALES

The Plectascales are a very heterogeneous group, including fungi in which the asci are distributed irregularly within small, spherical cleistocarps, and others in which the asci are formed separately, without association together in a fructification.

Penicillium, Link

Conidiophores erect, irregularly penicillately branched; conidia catenulate, hyaline or clear-coloured.

Cleistocarps small, spherical, generally brownish, rarely, or, in some species, never, produced. Sclerotia, which simulate cleistocarps, occur in some species.

The genus has been monographed by Thom [24] and by Biourge [25]. Many of the species can only be distinguished by their cultural behaviour. To this genus belong some of the commonest moulds, most of which are saprophytic. The following species are sometimes parasitic.

Penicillium expansum, Thom

This green mould is one of the commonest causes of rotting of apples in storage, as pointed out by Kidd and Beaumont [26]. Its spores are almost constantly present in the air, and frequently occur, therefore, on the surface of apples. The germ-tubes penetrate the flesh of the apple by way of abrasions in the skin, through the 'eye' or remains of the stalk, and through the lenticels. Enzymes secreted by the mycelium cause a soft brown rot of the fruit. The conidiophores are often concrescent, forming coremia. The fungus may grow down from the 'eye' into the core, sometimes setting up a rot in the interior, when apples are kept under very humid conditions. The relative humidity of the air is also an important factor in determining whether infection occurs through the lenticels.

Penicillium italicum, Wehmer

This is the common bluish mould of rotten oranges and other citrus fruits. The fungus enters the fruit through wounds, which are usually minute; it can also spread to healthy fruits by contact with diseased ones. The incidence of the rot increases as the fruit matures. In consequence of the greater care now taken to avoid wounding the fruit during picking

and packing, less rot is caused by this fungus than hitherto. Investigations in the United States by Fulton and Bowman [27] and by Barger and Hawkins [28] have shown that by immersing the fruits on gathering in a $2\frac{1}{2}$ to 5 per cent. solution of borax at 120° F. for 5 mins. losses from this fungus are greatly reduced.

Penicillium digitatum, Sacc.

The colour of this species is olive-green. It also causes a rot of citrus fruits, especially lemons. Its mode of life on these fruits is similar to that of the previous species, although it spreads less by contact.

Penicillium Gladioli, McCulloch and Thom

According to McCulloch and Thom [28a] this species causes a dry rot of Gladiolus corms, particularly during storage. The lesions are reddish-brown, sunken, and usually irregular in shape and size. A. Smith * has investigated this disease in England. Infection occurs only through wounds or from the shrivelled remains of the old corms. The fungus may perhaps grow as a saprophyte in the soil. This species produces small, pinkish-buff sclerotia as well as bluish-green conidiophores, the sclerotia being often seen on diseased corms. The fungus develops most readily on corms stored under humid conditions. Upon being lifted the corms should be well dried and stored in a cool, dry atmosphere ; it is advisable to remove the remains of the old corms. This species does not affect tulip or narcissus bulbs.

Penicillium Narcissi, A. Smith

This species attacks Narcissus bulbs and is sometimes responsible for large losses during storage.* Partially diseased bulbs, which look sound externally, are often planted, and this is one of the ways in which the disease is propagated. Infection also occurs through wounds or by the downward growth of the fungus from the dead remains of the top of the scale

* Work recently done at Cambridge by Dr. A. Smith, but not yet published.

leaves. The disease is usually at first confined to the scale leaf originally attacked, the fungus not passing laterally into other scales until the base is reached. Care should be taken to dry the bulbs well on lifting and to store them in a dry atmosphere. Only sound bulbs should be planted. The conidiophores of this fungus are bluish-green in mass; sclerotia are not formed.

Thielavia, Zopf

Conidia * of two kinds: (*a*) brown, formed in chains as chlamydospores; (*b*) hyaline, formed 'endogenously' in series from conidiophores.

Cleistocarps (perithecia) small, spherical, brown, closed, without appendages.

Thielavia basicola, (B. & Br.) Zopf

Chlamydospores * separating at maturity, 12×5–$8\,\mu$; hyaline conidia * 10–20×4–$5\,\mu$. Cleistocarps (perithecia) 80–$100\,\mu$; asci oval, 8-spored; ascospores lenticular, brown, unicellular, 10–$13 \times 4 \cdot 5$–$6 \cdot 5\,\mu$.

This fungus causes a root disease of many cultivated plants (lupin, pea, violet, &c.), and is particularly serious on tobacco in Kentucky and Connecticut. It became almost the limiting factor in the successful cultivation of tobacco in Ireland, and is very troublesome in Italy. It is, however, sometimes only a secondary organism, and some serious diseases have been wrongly attributed to it.

A part or the whole of the root system may be attacked by this parasite; in tobacco, a new group of roots may be formed at the crown above the injured region. In the early stages of attack a whitish fungus may be seen growing from the diseased roots, which later becomes dark-coloured. An affected plant may be killed outright, but, more frequently, its growth is stunted. Johnson and Hartman [30] state that the fungus is favoured by comparatively low soil temperatures; affected plants sometimes recover when the temperature rises.

In tobacco there are striking differences in varietal susceptibility; the disease in this crop can be largely controlled by

* Recent work by McCormick (29) indicates that these spores probably belong to a distinct fungus, *Thielaviopsis basicola*, (Berk.) Ferraris.

the use of resistant varieties and by a 4-year rotation. The disease is favoured by lime in the soil.

Plectodiscella, Woronichin

Stroma sub-epidermal in leaves and stems, containing somewhat scattered, globose asci, each containing 8 four-celled ascospores. The systematic position of this genus is uncertain. In Gäumann's *Vergleichende Morphologie der Pilze* it is placed in the Myrangiales.

Fig. 20. *Plectodiscella veneta*, lesions on raspberry cane ('Baumforth's Seedling'). Natural size.

Plectodiscella veneta, Burkholder   Raspberry Anthracnose or Cane Spot.

Ascospores ovoid-elliptical, 3-septate, hyaline, $12–21 \times 6 \cdot 5–8\,\mu$.

The conidial stage is *Glocosporium venetum*, Speg.; conidia oblong to elliptical, unicellular, hyaline, $5–7 \times 2 \cdot 5–3\,\mu$.

Raspberry anthracnose occurs commonly in the United States, where it has been investigated by Burkholder [31], and also in Britain, where it has been described by Harris.* This anthracnose occurs occasionally on the loganberry. The disease appears in the spring as dark purplish spots on the young canes, which increase in size, becoming grey in the centre. Similar spots appear on the leaves, which may become curled. The lesions on the stem are usually superficial, but, when deeper, form canker-like wounds. Where the spots are close together, the upper parts of the canes may die back; canes affected by anthracnose are usually shorter than healthy ones. Conidia are formed on acervuli in the lesions. Late infections on the young canes appear in the winter as white spots with minute, black dots, which are incipient acervuli. According to Burkholder [31], ascocarps begin to develop in the late summer in the stem lesions, but do not liberate the asco-spores until the spring. The fungus usually overwinters, however, as mycelium in the canes, which forms fresh acervuli in the spring.

The raspberry variety ' Baumforth B ' is most susceptible to this disease in England. Care should be taken to use only healthy canes when establishing new plantations. R. V. Harris (of the East Malling Research Station) states that perhaps the disease may be reduced appreciably by spraying with lime-sulphur when the buds open, and again after flowering.

Nematospora, Peglion

Vegetative phase yeast-like or hyphal; asci (sometimes called ' sporangia ') not grouped together in fructifications, cylindrical, containing 6–12 spores in two bundles; spores filiform, hyaline, each with a long appendage.

No adequate diagnosis of this genus has yet been published. It probably belongs to the Saccharomycetaceae, a family of the Plectascales.

Nematospora Coryli, Peglion

Peglion [32] first described this fungus as the cause of a mal-formation of hazel nuts in Italy, but the same species and

* Fifteenth Annual Report, East Malling Research Station, Kent, 1928.

another closely related to it have been recently found by Nowell [33] to be responsible for a serious internal boll disease of cotton in the West Indies. The second species, which has been studied by Marsh [34], also causes a similar disease of cotton bolls in Africa.

The cotton disease is transmitted entirely by insects, chiefly the green and stainer bugs, which, upon puncturing the boll, infect the seeds and the lint, resulting in partial or complete destruction of the seeds, discoloration and shattering of the lint, and sometimes the premature fall of the boll. Outwardly the boll often appears uninjured. In the West Indies the later part of the crop is most liable to injury owing to the greater abundance of stainers then. By eradicating other plants on which the insect carriers breed, the disease has been greatly reduced in St. Vincent.

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FUNGUS DISEASES (*continued*): PEZIZALES, HELVELLALES, PHACIDIALES, HYSTERIALES

PEZIZALES

APOTHECIA cup-like or disk-like, closed at first but open at maturity, the inner surface being covered with a hymenium of asci and paraphyses. Conidia and sclerotia frequently occur.

Dasyscypha, Fries

Apothecia small, short-stalked, brightly coloured in the disk, hairy on the outside ; asci cylindrical or clavate, 8-spored ; ascospores elliptical or fusiform, hyaline, unicellular or rarely 2-celled ; paraphyses filiform.

Dasyscypha calycina, (Schum.) Fckl. Larch Canker or Larch Blister.

Apothecia cup-like or disk-like, 2–3 mm. broad, short-stalked, yellowish without, orange within ; asci $140–200 \times 9–15\,\mu$; ascospores $17–23 \times 8–10\,\mu$, with rounded ends, unicellular, hyaline, uniseriate.

Hiley[1] states that the dimensions of the fungus growing parasitically are larger than when growing saprophytically.

Conidia (? spermatia) are formed in small pycnidia (? spermogonia), but are apparently functionless.

This fungus occurs abundantly as a saprophyte on dead larch twigs, and it is also a dangerous parasite of the European larch (*Larix europaea*), frequently causing the formation of large cankers. The disease is also serious on the Western American larch (*L. occidentalis*), but it affects the Japanese larch (*L. leptolepis*) only slightly.

Owing to the abundance of the fungus as a saprophyte, spores are commonly present in the air, but these can only cause infection of the living tree through wounds or dead tissues, on which the fungus first lives as a saprophyte. There has been much dispute as to the manner in which infection is

usually brought about. Wounds caused by mechanical injury, insects (the larch moth and *Chermes abietis*), and by frost, buds killed by shading of the upper branches, and dead lateral branches all afford means of entry. Hiley [1] considers that the latter are chiefly responsible for the formation of serious cankers in the main stem.

Once infection has begun, the mycelium invades chiefly the bark, although it also often enters the wood. The bark cells in the immediate vicinity of the fungus are killed by secretions, the mycelium advancing more rapidly in winter than in summer. Resin is deposited copiously in the dead cells and frequently exudes from the bark. On the borders of the invaded bark the tree reacts to the fungus by forming a cork layer in spring, which temporarily stays the progress of the parasite. Often, however, the fungus quickly evades the cork barrier and attacks a new zone of bark, after which another cork layer is formed, and so on. If the mycelium reaches the cambium, cork layers are formed, one after the other, radially across the bark year by year after successive invasions by the fungus, unless the progress of the fungus is permanently stopped. In this way a large canker is formed. The host tissue on the periphery of the canker and on the side of the stem opposite the canker is often swollen; for this reason foresters sometimes speak of the disease as larch 'blister'. Once the mycelium has reached the cambium the wood can be invaded; the hyphae penetrate both medullary rays and tracheids, making more rapid progress in the heart wood than in the sap wood in consequence of the higher air content of the former. The hyphae pass through the tracheid walls by minute holes. Such wood becomes reddish-brown in colour owing to the deposition of gum in the cells, but otherwise the wood is little changed. Where the cambium is killed at the

FIG. 21. *Dasyscypha calycina*, ascus and paraphysis.   × 250. (*R. W. Marsh.*)

base of a canker no more wood can be formed there, but the living cambium close by forms wood of an abnormal kind, which resembles 'wound-wood'.
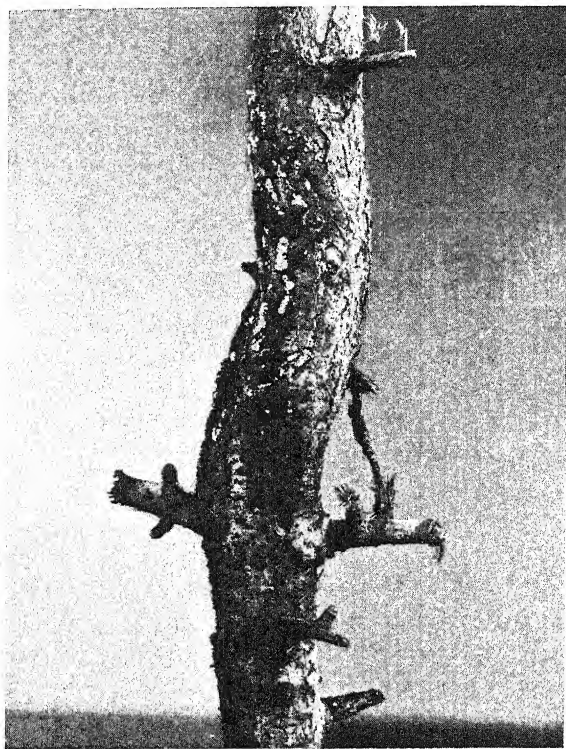
Fig. 22. Larch canker with apothecia of *Dasyscypha calycina*. ½ natural size.
(*W. J. Dowson.*)

Cankers are formed both on the main stem and on the lateral branches, but by far the most serious are those on the former. Hiley[1] points out that cankers on the main stem often arise a foot or two above soil-level, at the place of attachment of a lateral branch which has been shaded out. Such a branch is often affected by *D. calycina* growing sapro-phytically, and, by evading the cork barrier normally formed across the base of the branch, the fungus invades the main

stem. The remains of these laterals are often seen in cankers on the stem.

The most serious period in the life of the larch as regards this disease is from the third to the sixth year. The establishment of disease in the main stem then cripples the trees as regards timber-production, and may occasionally kill the trees outright. When the tree is older and the bark thicker, it is more difficult for serious cankers to be formed by the ingrowth of the fungus from dead, lateral branches.

The severity of the disease varies greatly in different areas. In its home-land, the mountains of Central Europe, *Larix europaea* is comparatively unaffected by Canker. In Britain, however, the disease is often serious, and many plantations have been ruined by it. The larch requires a good, well-drained, loamy soil, an open situation, and adequate rainfall for active growth. Where these conditions obtain in Britain the larch thrives. When growing vigorously, cork barriers are rapidly formed in the tissues in response to the fungus, which permanently prevent the parasite from developing seriously. With weak trees on the other hand the cork layers are too slowly or too imperfectly formed to check the fungus. Pure larch plantations are often more seriously attacked by this disease than mixed woods, probably, as Forbes [2] points out, because the surface root system of the larch has more scope for development in the latter than in the former.

Sclerotinia, Fuckel

Apothecia cup-like or disk-like, stalked, the stalk arising from a black sclerotium ; asci 8-spored ; ascospores elliptical or fusiform, unicellular, hyaline ; paraphyses thread-like.

In many species there are conidial forms and microconidia. The latter are very small, are often formed in chains, and only germinate with difficulty. The conidiophores are of diverse types, including Botrytis and Monilia forms. In some species conidia are produced much more abundantly than ascospores, and it is probable that some species of Botrytis and Sclerotium, in which an ascus stage either does not occur or has not yet been found, are related to this genus. There is great similarity between the microconidia of these forms and those of typical species of Sclerotinia.

Sclerotinia Sclerotiorum, (Lib.) de Bary (= *S. Libertiana*,
(Lib.) Fckl.)

Sclerotia large, up to 2 cm. long, usually about 1 cm., irregular in
form, though often roundish, black; apothecia arising in groups
from the sclerotia at the end of long, slender stalks, disk-like,
pinkish-buff in colour, 4–8 mm. across; asci cylindrical, 130–
135×8–$10\,\mu$; ascospores 9–13×4–$6\,\mu$; paraphyses clavate.
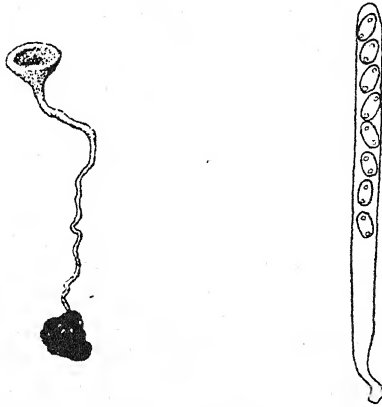
Fig. 23. *Sclerotinia Sclerotiorum*, (left) apothecium arising from a sclerotium,
natural size; (right) ascus, × 400.   (*R. W. Marsh.*)

There is hardly any destructive fungus that attacks such
a diversity of host plants as *S. Sclerotiorum*. It is the cause
of serious diseases of carrots, mangolds, potatoes, artichokes,
lettuce, beans, tomatoes, Antirrhinums, and other plants. As
de Bary [3] first pointed out, this fungus is generally unable to
penetrate healthy, uninjured tissues provided with a cuticle
or cork, but it often begins to grow on dead or moribund cells
and passes thence to sound tissues, which it rapidly destroys,
producing a soft rot. Its destructive action is due to the
secretion of toxic substances (probably enzymic) from the tips
of the hyphae, which, diffusing in advance, kill the protoplasm
and soften the cell walls, thereby affording further food for
the fungus.

The mycelium is white in mass and is sometimes seen on
the surface of diseased tissues. It rapidly forms sclerotia
on the surface of the host or in cavities within it. Upon

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disintegration of the tissues the sclerotia pass into the soil, where they remain dormant for months or sometimes years before germinating. The sclerotia only germinate when near the surface of the ground, the length of the stalks of the apothecia being such that the disks are just above soil-level. The spores are violently discharged from the asci, and if they alight on dead or moribund plant tissue they form mycelia.
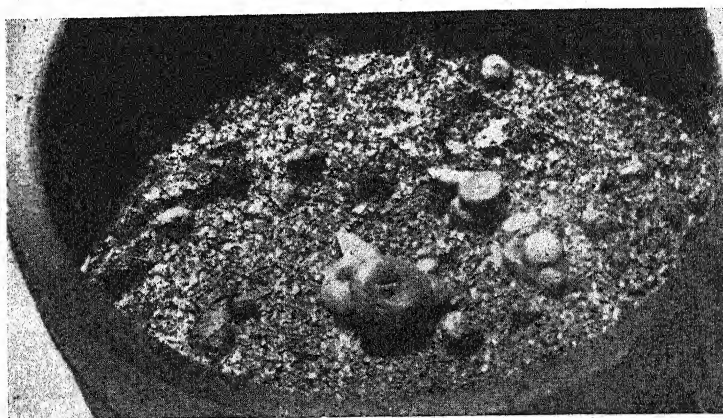
FIG. 24. *Sclerotinia Sclerotiorum*, apothecia at surface of soil in a flower-pot, which have developed from buried sclerotia.   Natural size.   (*W. J. Dowson.*)

From such a start the fungus becomes parasitic if opportunity offers. The sclerotia may also form mycelial strands on germination, by means of which infection through the soil is sometimes brought about. There is no specialization of parasitism in this fungus.

S. Sclerotiorum sometimes causes a serious rot of carrots, mangolds, and other roots in storage. The fungus may have already begun to infect a few of the roots before storage, and it may spread rapidly under the close conditions of the clamp. Badly wounded roots and any roots showing an incipient rot should not be stored.

Pethybridge [4] states that *S. Sclerotiorum* causes a serious disease of potato haulm in the west of Ireland, where it is known as 'stalk' disease. The spores first attack the yellowing leaves, whence the fungus passes into the stems, which gradually

collapse. Large sclerotia are formed in the pith cavity. These pass into the soil, where they germinate during the following summer if near the surface. Late planting tends to reduce losses from this disease, though adequate time must be allowed for the growth of the crop in order to get the maximum yield. Field beans may also be affected in a somewhat similar way.

In the United States this fungus is very troublesome in lettuce growing, particularly under glass, as described by Stone and Smith[5], and also in the transit of vegetables to market, as indicated by Ramsey[6]. In England Dowson[7] has shown that it attacks the flowers and stems of Antirrhinums under glass, infection being brought about by the spores germinating on the stigmas and corollas. It is suggested that bumble bees carry the spores to the flowers.

All material containing sclerotia should be burnt or buried deeply. In lettuce cultivation under glass, contaminated soil can be disinfected by steam or, according to Krout[8], by the use of formaldehyde at a strength of 1 in 50.

Sclerotinia minor, Jagger

This is closely related to the previous species, and, according to Jagger[9], causes a similar rot of lettuce in greenhouses in the United States. The sclerotia, however, are only about the size of mustard seeds, and upon germination may form mycelia that bring about infection through the soil.

Sclerotinia intermedia, Ramsey

According to Ramsey[10] this fungus is more or less intermediate between the two previous species, the sclerotia being 1–3 mm. in diameter. The mycelium is olive-buff in colour in contrast to the white mycelium of *S. Sclerotiorum*. In the United States it causes a rot of salsify and carrots while being marketed.

Sclerotinia Trifoliorum, Erikss. Clover Rot.

Sclerotia smaller than in *S. Sclerotiorum*, about 5 mm. across, irregularly spherical or somewhat flattened; apothecia as in *S. Sclerotiorum*; asci $180 \times 12\,\mu$; ascospores 14–$16 \times 6\,\mu$.

This species also is closely related to *S. Sclerotiorum*, but it affects a narrower range of hosts and the sclerotia germinate chiefly in the autumn. It causes a serious disease of red

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clover and other leguminous fodder plants in some parts of
England and other countries. One of the popular names for
this disease is 'Clover Sickness', but as there is an eelworm
disease called by this name, it is preferable to speak of the
fungus disease as 'Clover Rot'.

In October and November clover, which is usually sown
with the previous barley crop in the eastern counties of
England, often becomes rotten in patches in the field. The
outer leaves, especially those in a moribund state, are first
affected; the fungus then passes to the green leaves and
crown, causing them to become brownish-black and rotten.
At first only plants here and there in the field are affected,
but in wet, warmish weather the fungus spreads centrifugally
so that large groups of plants are killed, leaving bare patches.
Under suitable weather conditions the fungus may kill prac-
tically the whole field, but if a cold or dry spell intervenes
the disease does not develop further for the time being. In
early spring, however, the disease may again spread actively.
If plants affected in the autumn are not killed outright they
resume vigorous growth again in the spring in favourable
weather.

Upon the rootstocks of the dead plants black sclerotia are
formed. These remain dormant until the following autumn or
for several years if they are deeply buried. Germination pro-
ceeds as in *S. Sclerotiorum*, the apothecia being pinkish-buff in
colour. The ascospores are carried by wind to neighbouring
clover leys, and cause infection if the weather is suitable.

If the clover is not attacked during the first year it is
practically never affected in the second year of growth, and it
is a curious fact that clover growing spontaneously in mixed
herbage is not attacked by this disease. Clover sown with
rye grass may, however, be badly affected.

The disease has been studied in England by Amos[11] and
Wadham[12]. The former has found that the progress of the
fungus is sometimes impeded by allowing sheep to eat off the
clover in the autumn or winter if the foliage is luxuriant.
This tends to check the growth of the fungus from plant to
plant.

All kinds of clover except white clover may be seriously affected, but common red clover is most often attacked. Trefoil and sainfoin are less often troubled by this disease, and where ' Clover Rot ' is rife and the land suitable, one or other of these crops should wholly or in part replace clover. The disease is probably checked to some extent by extending the period between two clover crops, but this consideration is of weightier importance with the eelworm disease. In the eastern part of England it is usual to allow eight years between clover crops. Old clover leys should be ploughed in deeply, before October if possible, in order to prevent the sclerotia from germinating. The latter do not usually germinate before October.

Sclerotinia bulborum, (Wakker) Rehm

Sclerotia black, about 12 mm. across; disks of apothecia brownish, 3–5 mm. in diameter; asci $140 \times 9 \mu$; ascospores ovoid, $16 \times 8 \mu$.

Wakker [13] has investigated a disease of hyacinths, Scilla, and Crocus caused by this species, which is essentially the same morphologically as *S. Trifoliorum.* Cross inoculations show, however, that the hosts of the two species are different, and while the sclerotia of *S. bulborum* germinate mostly in the spring, those of *S. Trifoliorum* germinate in the autumn. In hyacinths the disease manifests itself after flowering by the leaves turning yellow and hanging down, the leaf bases being entirely destroyed. Besides forming apothecia the sclerotia simultaneously form mycelial strands which pass into the soil and infect the bulbs. According to Wakker [13] this is the chief mode of infection, the ascospores only occasionally causing infection by germinating on the scale leaves. Wakker states that although the plants first attacked in a field are widely scattered, others near these soon succumb through infection by mycelial strands in the soil. The sclerotia are formed either between the scales or on the surface of the bulb or corm. Slightly diseased bulbs may escape detection. Some of the sclerotia pass into the soil.

In Holland the disease is most serious in Scilla, but hyacinths are most commonly attacked. Wakker [13] advocates

the immediate destruction of affected plants and the removal of the soil around them in order to prevent further contamination by the sclerotia.

Sclerotinia tuberosa, Fckl.

Wood anemones and other species of anemone are attacked by this fungus, which is closely related to the previous species. Very large black sclerotia are formed on the rhizomes. The apothecia are larger than in the previous species.

Sclerotinia candolleana, (Lév.) Fckl.

Wilson and Waldie [14] record the epidemic occurrence of this species in Great Britain during 1926 on the leaves of *Quercus pedunculata* and *Q. sessiliflora*. Circular, yellowish spots appear on the leaves towards the end of June; these turn brown and extend until most of the leaf becomes discoloured, although the diseased tissues sometimes fall away, leaving holes. After the leaves fall to the ground, black sclerotia, about 4 mm. across, develop in them. In the spring each sclerotium produces a brown, stalked apothecium about 4 mm. in diameter. The ascopores are elliptical and measure $7-9 \times 3-4 \mu$. In other countries the fungus has been found also on *Castanea sativa*.

Sclerotinia Ricini, Godfrey

Sclerotia black, elongate, irregular, 1–25 mm. long, usually 3–9 mm.; apothecia one to several from a sclerotium, usually about 6–15 mm. high, disk saucer-shaped when expanded, 1·5–4 mm. across; asci $80-100 \times 8 \mu$; ascospores hyaline, ellipsoidal, $9-12 \times 4-5 \mu$.

Conidiophores of the Botrytis type; conidia globose, hyaline, $7-10 \mu$, compactly grouped.

Microconidia globose, hyaline, $2-3 \cdot 5 \mu$, catenulate.

A disease of the castor oil plant in the United States, caused by this fungus, has been investigated by Godfrey [15]. The inflorescence is most commonly affected, the spores directly infecting the tender tissues under warm, humid conditions. The fungus may also attack the stems and leaves, but usually only when these are young or when infected bits of inflorescence fall on them. Diseased tissues rapidly become rotten and produce masses of conidiophores of the Botrytis type, the spores being spread by insects as well as by wind. Young fruits also may be attacked, the seeds becoming wholly or

partly affected. Seed which is slightly contaminated may be the means of distributing the disease. Black sclerotia are formed in the dead parts of the host; these remain embedded in the tissues or pass into the soil, and produce apothecia on germination.

Godfrey [15] has shown by pure culture methods that the apothecial and conidial stages belong to one fungus.

The disease is only epidemic under extremely wet conditions. It has not yet been recorded from India or Central Africa, where the crop is grown on a large scale.

Sclerotinia Geranii, Seaver and Horne

Seaver and Horne [16] have shown by modern methods that in this species also a Botrytis stage is formed in cultures from the ascospores. The fungus affects the rootstocks of *Geranium maculatum* in the United States.

Sclerotinia Fuckeliana, de Bary   Grey Mould.

Sclerotia black, very irregular in shape, variable in size, but usually smaller and thinner than those of *S. Sclerotiorum*, often with portions of the host tissue more or less embedded; apothecia arising in groups of two to three from the sclerotium, stalk 5–10 mm. long, disk light brown, $\frac{1}{2}$–3 mm. across, asci 130×12–13μ, ascospores uniseriate, ovoid, hyaline, 10–11×6–7μ.

Conidial stage = *Botrytis cinerea*, Pers., forming dense grey tufts becoming brownish, ultimate branches of conidiophores with closely aggregated conidia, each at the end of a minute sterigma; conidia variable in shape and size according to the strain, often subglobose or oval, about 10–12μ in diameter.

'Attachment organs', consisting of closely branching systems of hyphae becoming blackish in colour, are often formed when the mycelium comes in contact with some resistant substance; there is every intergrade between a simple type of attachment organ and a sclerotium.

Fig. 25. Conidiophore of *Botrytis cinerea*, × 175. (*R. W. Marsh.*)

There has been much dispute as to whether *Botrytis cinerea* is a stage in the life-history of a Sclerotinia, and there is still no adequate proof of this by modern methods. de Bary [3] was of the

opinion that it was the conidial stage of *S. Fuckeliana*, and Istvánffi [17] found that sclerotia of the Grey Mould of the vine formed apothecia under some conditions and conidiophores of *B. cinerea* under others. It has been suggested that Istvánffi was dealing perhaps with two fungi, one a Botrytis and the other a Sclerotinia, but in the writer's opinion this is unlikely. Under British conditions sclerotia of *B. cinerea* invariably produce conidiophores on germination. Many strains of *B. cinerea* may have lost the power of forming apothecia. As *B. cinerea* frequently forms microconidia, which are practically identical with those of species of Sclerotinia, *B. cinerea* probably belongs to this genus.

In view also of the close pathological similarity between *B. cinerea* and other species of Botrytis on the one hand and species of Sclerotinia on the other, it is most convenient to describe these forms here. The fact that *S. Ricini* and *S. Geranii* have been shown recently to possess Botrytis stages, similar to *B. cinerea*, is additional confirmation of the writer's viewpoint.

Included in *B. cinerea* are many different forms or strains, many of which, genetically, are of the nature of 'elementary species', as their characters are sufficiently constant. Many of these forms are of omnivorous habit, though some are more specific in their choice of host. Some have recently been raised to the rank of species.

The parasitism of *S. Fuckeliana* (*Botrytis cinerea*) was first investigated in detail by de Bary [3], who pointed out that, ordinarily, the ascospores and conidia were unable to infect healthy tissues directly except in the case of such delicate structures as petals, &c., but that if the fungus had grown saprophytically, even for a short time, healthy plant tissues could be invaded, the cells being killed a little in advance of the hyphae. Marshall Ward [18] isolated an enzyme from a Botrytis attacking lilies, which caused swelling and disintegration of cell walls, and Brown [19] has indicated that the enzyme which softens the cell walls is probably also the cause of death of the host cells. Blackman and Welsford [20] have observed in detail the mode of penetration of the epidermis by germ tubes when growing saprophytically, the cuticle being penetrated mechanically after formation of an appressorium. Brooks [21] demonstrated that the conidia, while incapable of infecting healthy green tissues provided with a cuticle, are able directly to infect yellowing leaves. In nature initial attack of moribund tissues is one of the

commonest ways in which the fungus becomes a destructive
parasite.

Very many crop plants, including vegetables, potatoes, bulbs,
tomatoes, lettuce, young shoots of conifers and other trees,
vines, and gooseberry bushes suffer from attack by *B. cinerea*.
Only under very humid conditions is the fungus particularly
destructive. Once the fungus has begun to invade healthy
tissues it may spread at an alarming rate. The tissues are
rotted and become covered with a dense growth of conidio-
phores. Black sclerotia are formed on and in the tissues,
which, after a period of rest, give rise to conidiophores, or,
according to de Bary[3] and Istvánffi[17], sometimes apothecia.

Herbaceous plants are commonly attacked by this fungus
under greenhouse conditions. A scale leaf, a yellowing leaf,
a bit of plant débris, or a small wound, may all afford the
necessary start for the invasion of sound tissues, and the
fungus may occasionally directly affect delicate organs poorly
provided with cuticular protection, such as petals and very
young leaves, when the atmosphere is humid. In tomato
cultivation under glass the fungus often establishes itself on
the main stem if the lower leaves are cut off during the later
stages of cropping and the greenhouse is badly ventilated.
Bulbs are often affected by *Botrytis cinerea*, though tulips
are most commonly attacked by the form now differentiated
as *B. Tulipae*. Slightly diseased bulbs which appear sound
may be planted, with the result that the bulb rots in the
ground or throws up shoots which are diseased. *B. Douglasii*,
which kills seedlings and young shoots of the Douglas fir and
other conifers, is probably only one of the innumerable forms
of *B. cinerea*. In wet seasons *B. cinerea* may damage potato
haulm considerably, as pointed out by Pethybridge[22]. In this
case the spores directly infect young healthy foliage as well
as yellowing leaves. Istvánffi[17] has studied the disease of the
vine caused by this fungus in Hungary. Not only are the
grapes affected in a wet season, but young shoots and the woody
parts of the vines are invaded by the mycelium. Brooks and
Bartlett[23] have investigated the serious die-back of gooseberry
bushes caused by this fungus. Infection is brought about

often by way of dead portions of the smaller twigs, and the fungus growing downwards, even in the old wood, attacks one branch after the other in the region of the collar until the whole bush dies. The disease is most conspicuous in May, when large branches wilt and turn brown. Embedded in the bark of dead branches or in the collar of the bush are sclerotia, from which dense tufts of conidiophores protrude. In this disease carelessness in picking or in hoeing between the bushes often produces wounds which may be a means of entry for the fungus. Snags left in pruning may enable the fungus to attack the bushes. This die-back disease of gooseberry bushes is most serious on poor or badly drained soils. *B. cinerea* often kills young rose shoots in spring, especially when the nights are frosty.

In general, diseases caused by *Botrytis cinerea* can only be prevented by growing the plants under conditions as favourable as possible, by allowing free circulation of air, and by the destruction of dead and moribund plant tissues.

Botrytis Tulipae, (Lib.) Lind. (= *B. parasitica*, Cav.) 'Fire' or Blight of Tulips.

Sclerotia black, small, 1–2 mm. in diameter, circular in outline, flattened ; branches of conidiophores arising at an angle of about 60°, dichotomous ; conidia large, 12–24 × 10–20 μ, obovate, reddish-brown in mass ; microconidia globose, 3 μ, formed in chains from special penicillate conidiophores.

This disease occurs wherever tulips are cultivated extensively. There has been much confusion in the nomenclature of this fungus, as pointed out by Hopkins [24], who has investigated the disease in the United States. The conidia can infect the leaves and flowers directly under cold, wet conditions, the germ tubes penetrating the tissues either through the stomata or between the epidermal cells without the formation of appressoria. The withering tips of leaves and other moribund parts may also be channels of infection. The mycelium is both inter- and intra-cellular. Foliage attack causes a severe blight, which is called 'Fire' in England because of the scorched appearance of the leaves. The fungus passes from the leaves to the

bulb, on or in which it forms sclerotia. Only a few of the scale leaves may be affected, and when the bulbs are lifted there may be no external sign of injury. The fungus remains more or less dormant during storage, but after planting the disease begins to extend, causing a complete rot or spreading to the green shoots on which conidia are formed.

Practically all varieties of tulips are susceptible, but the fungus does not affect other bulbs under natural conditions. On most culture media the fungus forms innumerable sclerotia to the almost entire exclusion of conidia, although the latter occur abundantly in partly dried plate cultures or when the fungus is grown on sterilized tulip leaves.

Some of the sclerotia may lie dormant in the soil for several years, although most of them germinate after the first winter; in some parts of Holland tulips cannot be grown on soil contaminated in this way. Diseased plants in the field should be destroyed promptly and the soil around them removed. Care should be taken to plant only healthy bulbs as far as possible.

B. cinerea also attacks tulips, but not so frequently as *B. Tulipae.* These two diseases of the bulbs can usually be distinguished by differences in the sclerotia.

Botrytis narcissicola, Klebahn   Smoulder of Narcissus.

This species, which is closely related to *B. Tulipae,* causes a serious disease of Narcissus bulbs in Holland and N. Germany. Westerdijk [25] states that infection of the leaves takes place at soil level; the leaves turn brown and wither, and the fungus grows down into the bulb, in which black sclerotia, about 2 mm. in diameter, are formed between both the inner and the outer scales. On germination the sclerotia give rise to Botrytis conidiophores. Slightly affected bulbs may appear sound externally, but if planted they either rot or send up yellowish leaves which quickly die. Dowson [26] states that this disease is not uncommon in England on imported Narcissus bulbs. The conidia of this species are narrower than in *B. Tulipae.*

Botrytis polyblastis, Dowson

Conidiophores stout, $\frac{1}{2}$–$1\frac{1}{2}$ mm. long, with very short branches at the ends; conidia few, pear- or balloon-shaped, attached by long, narrow sterigmata, 30–50 μ in diameter, producing 6–13 germ tubes.

Sclerotia elliptical, up to 6 mm. long. Microconidia similar to those of other species of Botrytis and Sclerotinia.

Dowson [26] has described this peculiar species of Botrytis, which is the cause of a disease of Narcissus leaves in S.W. England and N. Ireland. Large yellow blotches with a greyish centre appear, turn brown, and extend until the leaf is killed. Conidiophores are only sparsely produced, but sclerotia are formed in the dead tissues. The bulbs are weakened by the early destruction of foliage. The disease is most common during a wet spring.

Botrytis Allii, Munn   Neck Rot of Onions.

B. Allii is closely related to *B. cinerea*, but the conidiophores are very short (about 1 mm.) and frequently unbranched, and the conidia are elliptical, $7–16 \times 4–6\,\mu$. In cultures the aerial mycelium is very low, dense and matted.

Great losses of onions in storage both in America and Europe are caused by this fungus. According to Munn [28] the initiation of infection occurs almost invariably in the field. The fungus chiefly attacks the necks of the onions, either directly or through yellowing leaves, whence it passes down between the three or four outer scales of the bulb. In a wet harvest infection may take place through dying leaves while the bulbs are drying. If the onions are imperfectly dried or if they are stored under humid conditions the fungus spreads and rots the bulbs. The rot may become offensive through secondary bacterial infection. The base of the bulb is sometimes first attacked by infection from the soil. Sclerotia are frequently seen on the neck or surface of the bulb of onions in storage. The sclerotia form conidiophores on germination.

The inflorescences of seed onions may be severely attacked in a wet season, infection occurring through stigmas, anthers, and pedicels.

Onions previously attacked by *Peronospora Schleideni* are especially susceptible to neck-rot as they are generally soft and poorly matured. Onions with thick necks are most liable to the disease as they dry with difficulty. Late applications of fertilizers and applications of manures which tend to delay maturity should be avoided. The onions should be properly dried before storage, and the rooms in which they are kept should be dry, well-ventilated, and maintained at a low temperature.

Walker [29] states that two other species of Botrytis (*B. bys-soidea*, Walker, and *B. squamosa*, Walker) also cause neck-rots of onions; these can be distinguished from *B. Allii* by their growth on potato-dextrose agar. According to him coloured varieties are less susceptible to all three species than white varieties. Walker [30] has found that artificial curing of the bulbs, sufficient to desiccate the neck tissue within a fortnight of harvest, greatly reduces losses in storage. *B. cinerea* may also induce rotting of onions.

Botrytis Paeoniae, (Oudem.) van Beyma

The characteristic features of this Botrytis are the swellings at the base of the conidiophores, and the oblong or oblong-ovate shape of the conidia, which measure $10-22 \times 7-9\,\mu$ (average $13\cdot97 \times 8\cdot14\,\mu$); sclerotia small, about 1 mm. across.

The disease of paeonies and lily of the valley (*Convallaria majalis*) caused by this Botrytis was first described by Ritzema Bos [31]. The fungus has been fully described by van Beyma [32]. Paeony shoots appearing just above the ground in the spring are attacked at about soil level; they wilt, turn brown, and become covered with conidiophores. Later in the season the fully developed leaves may be infected. The rot may extend to the underground parts, in which small sclerotia are formed, but the plants are rarely killed outright. The sclerotia infect the soil, which should not then be used for growing paeonies for several years. In transplanting paeonies the remains of the leaves should be cut off as these may be already invaded by the fungus. *Botrytis cinerea* also sometimes attacks the upper parts of the shoots during a wet summer.

The leaves and aerial stems of *Convallaria majalis* are also attacked by *Botrytis Paeoniae*, but the rhizomes are not affected.

Sclerotium cepivorum, Berk. White Rot of Onions.

This fungus also is probably closely related to species of Sclerotinia and Botrytis as it possesses the same type of microconidia, which, however, have not yet been seen to germinate. As far as is known, the fungus is reproduced solely by means of small black sclerotia, about $\frac{1}{2}$ mm. across, which produce hyphae on germination.

S. cepivorum affects onions, garlic, and, to a lesser extent, shallots and leeks. The first sign of attack is wilting and yellowing of the foliage, but, before this is apparent, the roots have been more or less destroyed by infection from the soil. The fungus spreads rapidly round the bulb as a white fluffy mass, which may grow up the neck of the onion ; later, the mycelium enters the bulb, which gradually dries up. Innumerable minute, black sclerotia occur on the shrivelled bulbs. Sclerotia left in the soil may contaminate it for several years.

The disease is disseminated largely by transplantation of slightly diseased seed sets or seedlings, which carry sclerotia on them or in adhering particles of soil. Cotton and Owen [33] state that in England the disease occurs chiefly in market gardens and allotments. The disease is also known in Italy and the United States. Land on which the disease has occurred should not be used again for onions for a period of six to eight years.

Sclerotinia cinerea, Schröter   Blossom Wilt, Spur Blight, Wither Tip, and Brown Rot of Stone Fruits.

Apothecia arising from sclerotia embedded in mummified fruits, 0·5–3 cm. high, stalk dark brown, disk lighter, 5–15 mm. in diameter ; asci $125–215 \times 7–12 \mu$; ascospores hyaline, ellipsoidal, $7–19 \times 4–8 \mu$.

Conidiophores ($= Monilia\ cinerea$, Bon.) grey in mass, branched ; conidia catenulate, hyaline, ellipsoidal, very variable in size, $11–17 \times 8–13 \mu$; conidia produced on mummified fruits smaller than those on twigs and fresh fruits. Microconidia also are formed in cultures.

This fungus attacks plum trees and other stone fruits in several different ways, and is very harmful in certain seasons. In plum trees it causes a wilting and browning of the blossoms, a blight of the fruit spurs, cankers in the branches, a die-back of young shoots ('Wither-tip'), and a brown rot of the fruit. It has been exhaustively investigated in England by Wormald [34-36] and in New Zealand by Cunningham [37].

The first symptom of attack is a wilting of the expanding flowers due to infection by conidia or ascospores. The myce-

lium often passes into the fruit spurs through the pedicels, and these may be killed. It may advance from the spur into the branch causing a canker. When the attack is severe during prolonged dull weather almost the whole tree may appear brown in May owing to destruction of the foliage. Such an infestation is usually complicated by a bad attack of aphides, though the mutual relations between fungus and insect are not yet understood. The aphides may disseminate the spores. Later in the season the fruit may become infected by way of the stomata or lenticels, or through wounds, a brown rot being induced. The mode of stomatal and cuticular penetration of stone fruits has been described by Curtis [36a]. In fruit clusters the fungus frequently passes from one plum to another. Shoots of the current year may be killed back for a distance of 6–12 inches, this being known as the ' Wither-Tip ' form of the disease. Badly affected fruits become more or less mummified by the growth within of a thick-walled mycelium, which may be aggregated into sclerotia; such fruits remain on the trees during the winter.

The conidial pustules of the fungus are formed on the cankers, the dead fruit spurs, the young twigs affected by ' Wither-Tip ', and on the mummified fruits, from December until the summer. Pustules are also formed on the fruits as they rot. Apothecia arise in the spring from mummified fruits slightly buried in the ground, but, according to Wormald [38], these rarely occur in England. Cunningham [37] states that in New Zealand apothecia are only found where mummified fruits have been buried in hard, compact soil.

The disease is very variable in severity. A cold, wet spell in the spring following warmer weather that has stimulated the flower buds to open is especially favourable to the fungus. All commercial varieties of plums are susceptible, especially ' Czar ', ' Victoria ', ' Early Rivers ', and ' Monarch ', but the susceptibility of each varies with the season, partly in consequence of different times of flowering. In an epidemic attack practically the whole crop may be lost. Later in the summer rotting of the fruit is most severe in a wet season.

S. cinerea exists in at least two specialized races, the form

on plum and cherry (f. *pruni*) being unable to cause the blossom wilt of apples due to f. *mali*. The British form of the fungus on plums occurs on the Pacific coast of North America, but a somewhat different form more commonly exists in North America and New Zealand, which is distinguished as *S. americana*, (Wormald) Norton and Ezekial. This species chiefly attacks plum and peach fruits. *S. laxa*, Aderhold and Ruhland, is a closely related species on apricots, which should perhaps be included in *S. cinerea*.

S. cinerea f. *mali* causes a blossom wilt and canker of apple trees in England as described by Wormald[34], especially in the varieties 'Lord Derby', 'Cox's Orange Pippin', 'James Grieve', and 'Warner's King'. The same form may cause a blossom wilt of pears, particularly in the variety 'Fertility'.

Although these diseases are often very severe, little can be done at present to control them by spraying. In the winter and spring the pustules form fresh conidia over long periods so that little good is done by spraying unless the pustules are actually killed by the fungicide. According to Lees and Briton-Jones[39] there is some indication that spraying with a tar-oil wash in the winter reduces the disease. Good results have followed the excision of affected fruit spurs in apples, although it is hardly practicable to do this in large plum plantations after a bad attack. Mummified plums should, however, be destroyed. When large plum branches die back in consequence of combined attack with aphis, these should be cut out in June or early July in order to minimize the risk of infection of the wounds by *Stereum purpureum*.

Sclerotinia fructigena, Schröt. Brown Rot of Apples.

Apothecia similar to those of *S. cinerea*. Conidiophores (= *Monilia fructigena*, Pers.) buff-coloured in mass, otherwise as in *S. cinerea*; conidia 18–22×11–$13\,\mu$. The differently coloured and larger pustules, and the greater size of the conidia distinguish this fungus from *S. cinerea*.

This species causes a fruit rot of apples, pears, plums, and cherries, but occurs mostly on apples. Infection of the fruit generally takes place through small wounds, or through the
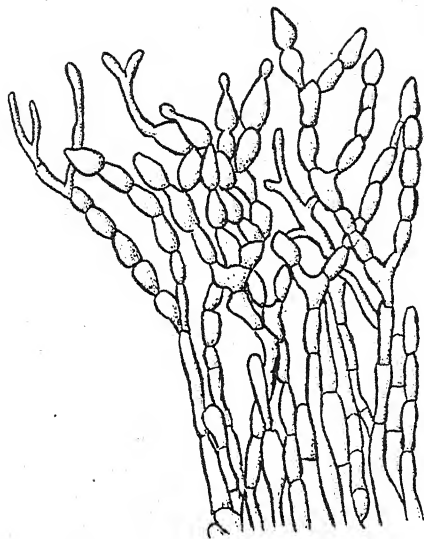
Fig. 26. Conidiophores of *Sclerotinia fructigena*, × 250. (*R. W. Marsh.*)
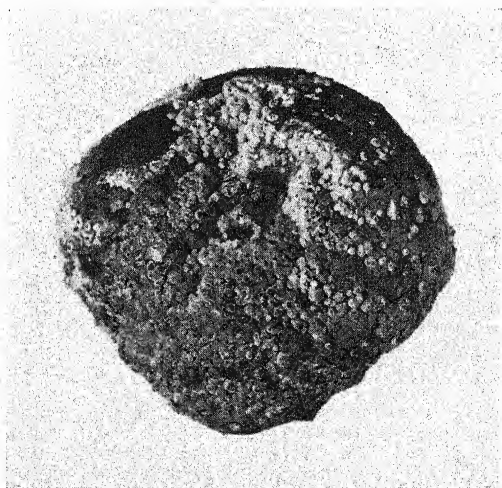
Fig. 26a. Conidial pustules of *Sclerotinia fructigena* on apple fruit.
Natural size.

L

lenticels, the flesh becoming brown and soft. Apples affected by brown rot usually fall from the tree. They may become mummified and give rise to new pustules in the following spring. In the United States and on the Continent mummified apples form apothecia in the spring if slightly buried. Dowson[40] has found that infection of apples on the tree may also take place through the eye, the mycelium passing down into the core, whence a rot begins. Such apples ripen and fall prematurely. The fungus occasionally passes from the fruit into the spurs, and thence into the branches.

S. fructigena is also responsible for a considerable amount of rot in apples during their early storage life, wounds made in picking and other abrasions having already become contaminated by spores before storage. Brown rot, however, does not spread secondarily in storage. Another form of rot caused by this fungus may affect stored apples. This results in a blackening of the surface accompanied by a brownish black rot inside; the skin and tissue immediately beneath it become a sclerotium. Such apples shrivel very slowly and do not bear pustules as do apples affected by the usual kind of brown rot. According to Spinks[41] the black form of rot chiefly depends on the nature of the variety, but Wormald[35] has shown that in some cases a distinct strain of the fungus is the cause of blackening.

Sclerotinia Mespili, Schellenberg, produces large, brown blotches on the leaves of the medlar, which smell sweetly, and on which spherical conidia, separated by disjunctors, are formed in chains. These spores infect the fruits, which become mummified. The latter give rise to apothecia in the spring, the ascospores infecting the leaves.

Sclerotinia padi, Wor. affects the leaves of *Castanea vesca* and *Prunus padus*, the conidia thereon infecting the young fruits. These become mummified and produce apothecia after the winter.

Other species of Sclerotinia

The life-history of *Sclerotinia oxycocci*, Wor. on the cranberry is essentially the same as that of the two previous species. *S. Vaccinii*, Wor. and *S. baccarum*, (Schröt.) Rehm, described by Woronin[42], on the cowberry and bilberry respectively, are similar.

S. Ledi, Naw. is heteroecious: the conidia on the leaves of
Vaccinium uliginosum infect the flowers of *Ledum palustre*, and
produce sclerotia which eventually form apothecia, the ascospores
infecting the leaves of the Vaccinium. According to Fischer [43]
S. Rhododendri is also heteroecious, alternating between the leaves
of the bilberry and the fruits of Alpine roses (*Rhododendron* spp.).
S. Ledi and *S. Rhododendri* occur on the Continent.
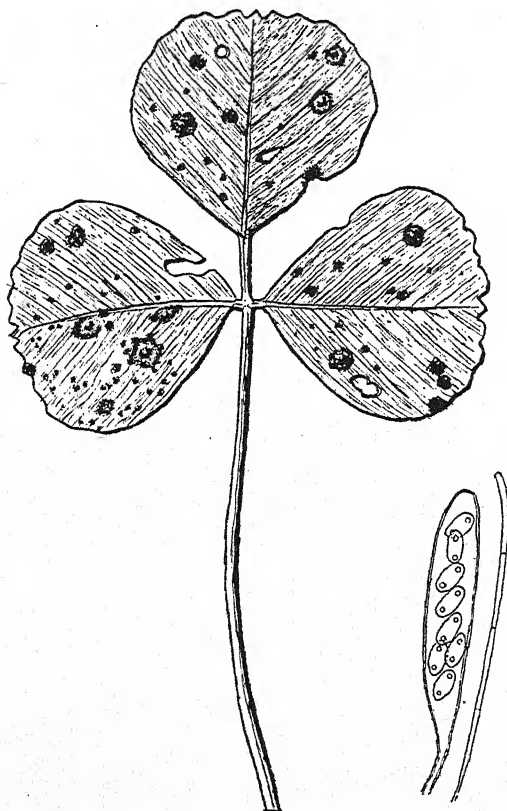
Fig. 27. *Pseudopeziza Trifolii*, (left) affected clover leaf, × 2; (right) ascus and
paraphysis, × 350. (*R. W. Marsh.*)

Pseudopeziza, Fuckel

Apothecia formed beneath the epidermis of host, finally erumpent;
asci containing 8 unicellular, hyaline spores; paraphyses some-
what stout.

L 2

Pseudopeziza Trifolii, (Biv.-Bern.) Fckl.

Apothecia in brown spots chiefly on the leaves, about 0·5 mm. across ; ascospores elliptical, $10–15 \times 4–6 \mu$.

Common red clover is often affected by this fungus, which produces small brown spots on the leaves, especially in the autumn and winter. Other kinds of clover are sometimes spotted in the same way. The disease is usually of no economic importance.

Pseudopeziza Medicaginis, (Lib.) Sacc., is hardly distinguishable from the preceding species except as regards host relationships. It affects lucerne (alfalfa) and black medick, causing considerable destruction of foliage in the United States in dry seasons.

Pseudopeziza Ribis, Klebahn   Currant and Gooseberry Leaf Spot.

Apothecia formed in spring on overwintered leaves.
    Conidial stage = *Gloeosporium Ribis*, (Lib.) Mont. & Desm. ; acervuli stromatic, conidia hyaline, $12–14 \times 5–9 \mu$.

The ascus stage of this fungus was first described by Klebahn [44]. The conidial stage commonly occurs in the summer on the leaves of red currants and to a lesser extent on black currants and gooseberries, producing numerous small, brown spots on the upper surface. The fungus sometimes causes premature defoliation.

Fabraea, Saccardo

This genus is similar to Pseudopeziza, but the apothecium develops on a covered subiculum, and the ascospores are 2- to 4-celled.

Fabraea maculata, Atk.   Quince Leaf Blight.

Apothecia formed on overwintered leaves ; ascospores 2-celled.
    Conidial stage = *Entomosporium maculatum*, Lév. ; acervuli black ; spores stalked, hyaline, $18–20 \times 12 \mu$, several-celled, the lateral cells small and attached to the median septum, each terminal cell with a long seta.

This fungus causes a blight of quince and pear leaves ; it also affects the fruit, causing spotting and cracking. The leaf spots are at first dull red with dark borders, and may become confluent. In severe attacks the leaves turn brown and fall

prematurely. The acervuli occur on the discoloured spots. The fungus is often serious in the United States, but is rarely harmful in England. It can be controlled by spraying with Bordeaux mixture.

Neofabraea, Jackson

As in Fabraea, but the apothecium breaks forth from a more or less exposed subiculum ; the ascospores are unicellular at first, but become 2- to 4-celled.

Neofabraea malicorticis, (Cord.) Jackson

This fungus causes a serious canker of apple trees in British Columbia and Oregon, and also a spotting and rot of the fruit, particularly in storage. Infection, which occurs chiefly in the autumn, may take place through uninjured bark, especially by way of the lenticels. The cankers are biennial, acervuli (*Gloeosporium malicorticis*) being produced in the first year, and apothecia in the following season. Apples imported into England from these regions are sometimes affected by a rot caused by this fungus. The conidia are curved and measure 15–$30 \times 3 \cdot 8$–$5 \, \mu$.

Zeller and Childs[45] point out that a closely similar disease of apple trees in Oregon is caused by *Glocosporium perennans*, but the cankers formed by this species are perennial.

Cenangium, Fries

Apothecia erumpent, leathery, brown or blackish, at first cup-like, then disk-shaped ; ascospores unicellular, hyaline.

Cenangium Abietis, (Pers.) Rehm

This fungus causes a die-back of pine twigs in Scotland, on the Continent, and in America, infection occurring through the buds. This disease has been very destructive to *Pinus austriaca* in Norway, as stated by Jørstad[46]. In addition to apothecia there are probably two pycnidial stages in the life-history, one with long, slightly curved, two- to five-septate spores (*Brunchorstia destruens*, Erikss. = *Excipulina pinea*, (Karst.) v. Höhn.), the other with small, unicellular spores. van Luijk[47], who states that *B. destruens* is very injurious to *P. laricio* var. *corsicana* in Holland, has described pure cultures of this fungus.

Dermatea, Fries

Apothecia arising from small stromata, leathery, black or brown, disk-like when expanded ; asci thick-walled with 8 to 4 spores ; ascospores at first unicellular, then 4- to 6-celled, hyaline or brownish ; paraphyses septate, brown.

Pycnidia often occur.

Dermatea Prunastri, (Pers.) Fries

Dowson [48] has described a die-back of branches of greengages caused by this fungus. It occasionally attacks other varieties of plums. The pycnidia (= *Sphaeronema spurium,* (Fr.) Sacc.) have long, protruding necks.

Other species of Dermatea are weakly parasitic on various kinds of trees.

Bulgaria, Fries

Apothecia large, dark-coloured, gelatinous when moist ; asci 4- to 8-spored ; ascospores unicellular, brown or hyaline.

Bulgaria polymorpha, Wett.

This fungus commonly grows on the bark of felled oak and beech. It may also attack and kill the bark of old trees, as described by Tabor and Barrett [49]. Invasion of the bark of living trees is accompanied by the exudation of a gum-like substance. The dead bark gives rise first to pycnidia, which exude black spore tendrils, and then to large black apothecia which are disk-like when expanded. Each ascus usually contains eight ascospores, of which four may be brown and four hyaline. The latter are usually smaller than the former.

Cyttaria, Berk.

Apothecia sunk in a more or less globular, erumpent stroma ; asci 8-spored ; ascospores hyaline, elliptical.

Pycnidia also occur.

Cyttaria Darwinii, Berk.

This genus was first described by Berkeley from material collected by C. Darwin on trees of a Nothofagus in Tierra del Fuego during his voyage in H.M.S. *Beagle,* the species being *C. Darwinii.* Other forms of Cyttaria occur on species of

Nothofagus in New Zealand and Tasmania. These fungi grow on the branches and stems of the trees, killing the tissues. The stromata bearing the apothecia may be as large as a small apple, and in *C. Darwinii* are bright yellow in colour.

Roesleria, Thum. & Pass.

Ascocarps small, stalked, the upper surface of the head bearing asci; asci cylindrical, 8-spored, the ascus walls rapidly disintegrating; ascospores unicellular, globose, hyaline.

The exact systematic position of this genus is uncertain.

Roesleria hypogaea, Thum. and Pass. (= *R. pallida,* (Pers.) Sacc.)

Ascocarps about 2 mm. high, head white, then brownish; ascospores 4–5 μ in diameter.

There is some doubt as to whether this fungus is parasitic. It occurs commonly on dead roots. According to Prillieux [50] it causes a serious root disease of vines in France, spreading through the soil. Marsh [51] inoculated the roots of roses and oaks with this fungus, but he found it entirely innocuous.

HELVELLALES

Fruit-bodies large and fleshy, but not cup-like, the hymenium being spread over the upper surface and exposed from an early stage.

Rhizina, Fries

Fruit-bodies sessile, expanded from the first, fleshy, the under surface bearing numerous tufts of hyphae; asci cylindrical, 8-spored; ascospores unicellular, hyaline.

Rhizina inflata, (Schäff.) Sacc. (= *R. undulata,* Fr.)

Fruit-bodies slightly convex, more or less circular, chestnut-brown; ascospores fusiform, $32–36 \times 9–10 \mu$, tips of paraphyses brown.

Although this fungus is sometimes only saprophytic, growing on woodland soil near tree stumps or on the sites of fires, it may be a dangerous root parasite of young conifers. It rarely attacks broad-leaved trees. There is danger of attack from this fungus when coniferous plantations are felled and

replanted with conifers, as pointed out by Brooks [52]. The fungus commencing to grow on the stumps or on the soil around them forms delicate mycelial strands, which invade the roots of the newly planted trees. When the mycelium reaches the collar, the young tree suddenly dies. The fructifications are formed as large, brown, crust-like masses on the soil around the dead trees.

PHACIDIALES

Apothecia more or less immersed, opening irregularly.

Phacidiella, Potebnia

Apothecium sunk in a stroma; asci cylindrical, 8-spored; ascospores uniseriate, elliptical, hyaline, with one or two large guttules; paraphyses filiform, violet above, covering the asci with an epithecium.

Pycnidium (= Phacidiopycnis, Potebnia) stroma-like, thick and blackish, pseudo-chambered above; pycnospores hyaline, of two kinds: microconidia rod-shaped, macroconidia ovate or nearly spherical.

Phacidiella discolor, (Mout. and Sacc.) Potebnia (= *Phacidium discolor*, Mout. and Sacc.).

Apothecia $\frac{2}{3}$–1 mm. across, brownish black, laciniate; asci 120–140 × 15–18 μ; ascospores ovate, 1–2 guttulate, hyaline, 17–22 × 8–10 μ.

Pycnidia (*Phacidiopycnis Malorum*, Potebnia) irregularly spherical or pear-shaped, greyish, becoming black, $\frac{3}{4}$–1 mm. across: microconidia rod-like, hyaline; macroconidia ovate, often pointed at the lower end, hyaline, with one large or several small guttules, 9–12 × 7–8 μ.

On certain culture media (e. g. Dox) the fungus buds profusely after the manner of *Dematium pullulans*.

Potebnia [53] considers *P. discolor* to be the cause of a canker and die-back of apple and pear branches in Russia. Southee and Brooks [54] have found the pycnidia of this fungus associated with a similar disease of apple-trees in England, and Brooks [55] has recently found apothecia also. The fungus appears to be only a weak parasite. Osterwalder [56] has described a rot of apples in storage caused by it, the skin becoming black and the flesh brown to brownish black.

Fuckelia conspicua, recorded by E. and E. Marchal [57] as causing a rot of apples and pears in Belgium, is the same fungus. Miss L. Solberg informs the author that *P. discolor* causes a black rot of stored apples in Norway, where it has been referred to hitherto as *Pyrenochaeta furfuracea*, (Fries) Rostrup.

Fig. 28. Disease of branches of ' Lord Derby ' apple caused by *Phacidiella discolor*. Natural size.

Cryptomyces, Grev.

Apothecia flat, black ; asci cylindrical, 8-spored ; ascospores unicellular, hyaline ; paraphyses thread-like.

Cryptomyces maximus, (Fr.) Rehm. produces long, black patches on the branches of willows, especially *Salix fragilis*, which are gradually killed. The ascospores are oval, obtuse, hyaline, 20–26 × 10–18 μ.

Keithia, Sacc.

Apothecia roundish; asci 4- or 2-spored; ascospores unequally 2-celled, brown.

Keithia thujina, Durand

Pethybridge [58] has found this fungus killing young trees of *Thuja gigantea* in Ireland; it is rather common in nurseries in the south of England. It also causes a serious leaf disease of *T. plicata* in the United States. The black fruit-bodies, which are gelatinous when moist, are formed on the leaves; the asci are two-spored.
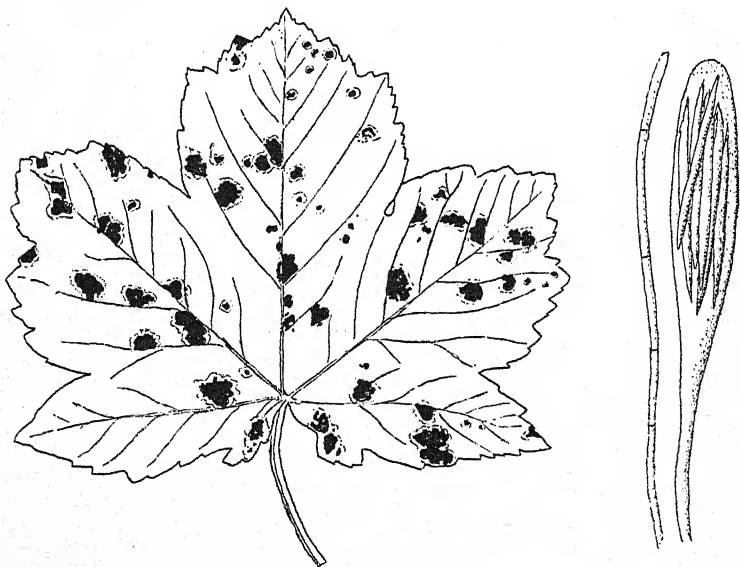
FIG. 29. (Left) Sycamore leaf affected by *Rhytisma acerinum*, $\frac{1}{2}$ natural size; (right) ascus and paraphysis, × 500. (*R. W. Marsh.*)

Rhytisma, Fries

Apothecia formed on a black, sclerotial stroma, opening irregularly by a lip-like slit; asci 8-spored; ascospores filiform, usually unicellular, hyaline; paraphyses filiform.

Rhytisma acerinum, (Pers.) Fr. Tar Spot Fungus.

Apothecia radiately arranged on the stroma, formed in over-wintered leaves; asci 120–130 × 9–10 μ; ascospores 65–80 × 2–3 μ.

Spermatia (= *Melasmia acerina*, Lév.) formed by the young stroma in the summer, minute, rod-like, hyaline, ungerminable as far as is known.

The black spots so commonly seen on the leaves of syca-mores and maples in late summer are caused by this fungus, but the disease has no serious effect on the trees. The black spots are the stromata of the fungus, which remain dormant on the fallen leaves during the winter. Re-infection takes place in the spring, the ascospores being ejected into the air from the stromata when the new foliage is developing.

Other species of the genus occur on the leaves of other trees, in some of which the ascospores are formed during the summer.

Rhabdocline, Sydow

Apothecia elongated, brown, breaking through the epidermis by a longitudinal slit; asci club-shaped, 8-spored; ascospores uni-cellular, cylindrical, hyaline.

Rhabdocline Pseudotsugae, Sydow, causes defoliation of the Douglas fir (*Pseudotsuga Douglasii*) in the north-western United States, and Wilson and Wilson [59] have found it injurious to the Douglas fir and the blue Douglas fir (*P. glauca*) in Great Britain. The fungus produces brownish patches in the leaves. The mycelium is very abundant in the affected tissues, and the cell contents become brown as the cells are killed. Infection occurs during the summer, and apothecia are formed on the under surface of the leaves in the following spring, the hymenium being orange in colour. The ascospores measure $17–21 \times 7–10\,\mu$ and become 2-celled and sur-rounded by mucilage after ejection.

HYSTERIALES

Ascocarps elongated, dehiscing by a narrow longitudinal slit.

Lophodermium, Chev.

Ascocarps black; asci 8-spored; ascospores thread-like or cylindrical, unicellular, hyaline. Pycnidia sometimes occur.

Lophodermium pinastri, (Schr.) Chev.

Ascocarps black, 1–2 mm. long; ascospores $90–120 \times 1.5\,\mu$. Pycno-spores unicellular, hyaline $6–8 \times 1\,\mu$.

This fungus is one of the causes of the shedding of pine needles ('pine leaf cast'), particularly in seed beds and in

young trees, although similar defoliation may also be due to drought or frost. Many species, including *Pinus sylvestris*, are liable to attack, but *P. strobus* is rarely affected. The fungus causes the formation of brown blotches on the leaves, which first give rise to minute black pycnidia, and then, frequently after the leaves are shed, to larger black ascocarps. The needles turn wholly brown before falling. Where the disease is prevalent it is advisable to establish pine nurseries in the midst of dicotyledonous woods or at some distance away from pine forests.

Several other species of Lophodermium attack coniferous leaves, sometimes causing defoliation. *L. macrosporum* is parasitic on the spruce, and *L. nervisequium* on the silver fir.

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FUNGUS DISEASES (*continued*): HYPOCREALES, DOTHIDEALES

HYPOCREALES

Perithecia bright-coloured, somewhat fleshy, asci with or without paraphyses, arising from the base. Conidial stages frequently occur.

Nectria, Fries

Perithecia isolated or grouped, somewhat soft, brightly coloured; asci 8-spored; ascospores 2-celled, usually hyaline. Conidia often present.

Nectria cinnabarina, (Tode) Fr. Coral Spot Fungus.

Perithecia aggregated, almost globose, red, about 0·5 mm. in diameter; asci $50–90 \times 7–12 \mu$; ascospores 2-celled, hyaline, $12–20 \times 4–6 \mu$.

Conidial pustules pink, 1–2 mm. across, covered with branching conidiophores; conidia hyaline, $4–6 \times 2 \mu$.

Although the Coral Spot fungus is most commonly saprophytic on dead twigs, it may be parasitic. It frequently causes a die-back of currant bushes, especially red currants, as described by Line[1], and it may invade wounds in trees such as sycamore, horse-chestnut, lime, fig, and apple. It also causes a die-back of the branches of tea-bushes in northern India. The fungus cannot infect healthy tissues, but readily enters currant-bushes through snags left in pruning and other wounds. These snags inevitably die, and the fungus, after growing in them, passes thence into healthy branches. The mycelium grows chiefly in the vessels, which become discoloured owing to the formation of gummy substances. The fungus spreads upwards and downwards, and when the whole of the wood at any position in a branch becomes permeated, the tissues above that region die. During the early summer large branches frequently wilt, and, if the collar of the bush is completely invaded, the whole bush succumbs. Old bushes

are much more liable to attack than young ones. The pink pustules of conidiophores are produced abundantly on the dead branches, and subsequently reddish perithecia, in groups, may be formed.
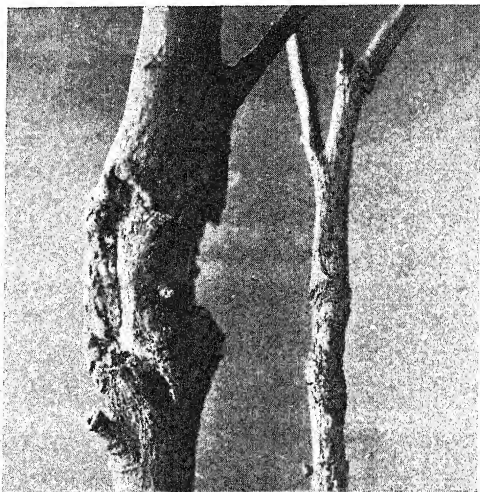
FIG. 30. Cankers in apple trees caused by *Nectria galligena*. ½ natural size. (*W. J. Dowson.*)

Nectria galligena, Bres. Apple and Pear Canker.

Perithecia almost globose, aggregated or scattered, dark red, about 0·5 mm. in diameter; asci $80-90\,\mu$; ascospores 2-celled, ovate-oblong, $12-16 \times 4-5\,\mu$.

Conidial pustules (= *Fusarium Willkommii*, Lindau) whitish or buff-coloured; conidia small and unicellular, or long, sickle-shaped, 1-5 septate, hyaline.

Cayley[2] has pointed out that the common cause of apple canker is this fungus, and not *N. ditissima*, Tul. (= *N. coccinea*, (Pers.) Fr.), as was formerly supposed.

In England this fungus causes one of the most serious diseases of apple- and pear-trees; it is widespread on the Continent, and it occurs commonly in North America, although it is not so dangerous there as in England. *N. galligena* also affects beech and willow trees.

This fungus kills young twigs and fruit spurs, and forms cankers in the branches, which may be so deep as to expose the wood. Conidial pustules are first produced, and are followed by small red perithecia. Both kinds of spores occur also on the fruits, the perithecia being not uncommon, according to Dillon Weston[3], on shrivelled apples remaining on the trees during winter. Dillon Weston[4] in England and Ferdinandsen[5] in Denmark have called attention to an eye-rot of ripening fruits due to this fungus. A blossom wilt of 'Worcester Pearmain' apples is also caused by it, according to Dillon Weston[4], and Harris[6] has observed canker in pear twigs initiated by infection of the leaf stalks, possibly through scab pustules (*Venturia pirina*).

Both conidia and ascospores cause infection through exposures in the bark, minute cracks such as those associated with an attack of woolly aphis, and leaf scars being sufficient for entry. The mycelium spreads chiefly in the bark, killing the tissues slowly as it progresses; it may extend into the wood. In old cankers the bark tissues on the periphery are considerably hypertrophied. Large cankers greatly impede the growth of the branches, and may completely girdle them. During the extension of the mycelium in the bark the host forms cork layers which temporarily check the fungus and which indirectly cause the formation of the concentric cracks conspicuous in young cankers.

Wiltshire[7, 8] has studied some of the chief modes of infection, and has shown that leaf scars are one of the principal channels of invasion. Spores alighting on cracks in the scars, soon after leaf-fall or in the spring, produce a mycelium which often penetrates the bark before a cork-barrier can be formed to impede it. In infections of this kind the adjacent buds are killed. Another common channel of infection is through young pustules of the Scab fungus (*Venturia inaequalis*) on the twigs. Cracks in the bark over the scab spot give lodgement to the canker spores in the autumn; the fungus then establishes itself on the stroma of the Scab fungus, and passes thence into the bark, unless the speedy formation of a cork layer below the scab pustule prevents this. Trees that

have been badly affected by Scab are often severely attacked shortly afterwards by Canker.

Varieties of apples and pears show great differences in susceptibility to this disease. Among apples, 'Cox's Orange Pippin', 'Lord Suffield', 'Stirling Castle', 'Warner's King', and 'Worcester Pearmain' are very susceptible, while 'Bramley's Seedling', 'Lane's Prince Albert', 'Newton Wonder', 'Blenheim Orange', and 'Sturmer Pippin' are usually resistant. The degree of resistance, however, depends greatly upon environmental conditions, especially soil factors. On heavy, poorly drained land, Canker is more severe than on well drained, loamy or light soil. A variety which cankers badly in one district may be little affected in another. Even the kind of stock on which a variety is grafted may influence the susceptibility to Canker. For instance, 'Bramley's Seedling', worked on 'French Paradise II', becomes so badly cankered that it often dies, whereas on other stocks it is usually very resistant.

In establishing new plantations of apples and pears the utmost care should be taken to select varieties of apples and pears that do not canker badly in the district. Poorly drained land should not be used for growing these fruits. Badly cankered branches should be cut out. A special degree of fruitfulness is sometimes associated with moderate attacks of Canker, as in the variety 'Stirling Castle'. This is probably due to partial ringing of the bark by the cankers. The economic life, however, of such trees is usually short. The Scab fungus should be kept under control by spraying; there is need for further investigation as to whether leaf-scar infection can be prevented by spraying in the autumn.

Nectria coccinea, (Pers.) Fr.

Westerdijk and Van Luijk [9] have pointed out that this species can be distinguished only with difficulty from *N. galligena*. It occurs commonly on the poplar, beech, elm, horse-chestnut, ash, and sycamore, causing either the formation of cankers or killing large areas of bark without producing cankers. Inoculation experiments indicate that it may give rise to cankers in apple-trees.

Nectria Rubi, Osterwalder

A disease of raspberry canes probably due to this fungus has been described by Osterwalder[10]. The foliage turns yellow before the affected canes die. The perithecia, which are purplish black when mature, are found chiefly on the underground parts of the canes. The fungus also has a Fusarium stage. This disease occurs in the British Isles and has been described by Pethybridge and Nattrass[11], but the parasitism of the fungus is still doubtful as successful inoculation experiments have not yet been carried out.

Nectria graminicola, Berk. and Br. (= *Calonectria graminicola*, (Berk. & Br.) Woll.)

This fungus grows as a white or reddish-grey mould on rye and wheat under snow in northern Europe, frequently causing a rot of the plants over large patches in the fields. In the mould stage the fungus produces a Fusarium type of spore (*F. nivale*, Caes.); the almost black perithecia develop on the dead plants. Eriksson[12] states that the fungus is carried over in the seed coats, and he advises sprinkling the seed with 0·1 per cent. corrosive sublimate before sowing.

Gibberella, Saccardo

Perithecia as in *Nectria*, but the hyaline ascospores are spindle- or sickle-shaped and 2- to 4-celled. Conidia usually of the Fusarium type.

Gibberella Saubinetii, (Mont.) Sacc.   Wheat Scab.

Perithecia bluish, $200–300 \times 170–220\,\mu$; asci $60–76 \times 10–12\,\mu$; ascospores fusiform, straight or curved, acute, 4-celled, $18–24 \times 4–5\,\mu$.

Conidia (= *Fusarium graminearum*, Schwabe) curved, acute, usually 5-septate, hyaline, $24–40 \times 4–5\,\mu$.

Mycelium effused, white to rose-coloured.

This fungus causes the death of young and older plants of wheat, maize, barley, rye, grasses, and occasionally other plants. Conidia of the Fusarium type are first produced on the dead tissues near soil level, followed by perithecia. The fungus also commonly affects the ears of cereals, causing

shrivelling of the grains, which are often covered with a pinkish mould. It is most destructive in the United States, but it occurs also in Europe and Australia. The disease is known as 'scab' or 'seedling blight' of cereals. The parasite is probably also one of several fungi causing 'foot-rot' in these crops. The 'scab' form of the disease on the grain is greatly favoured by wet weather.

Infection may be brought about through the use of infected seed, from stubble, or from the soil. Seedlings of wheat and maize are often killed either before or after emergence above the soil, or the plants may be merely stunted. The disease has been investigated by Atanasoff[13], and by MacInnes and Fogelman[14]. Dickson, Eckerson and Link[15] have studied its incidence in wheat and maize with respect to the temperature relationships of host and parasite. At relatively low soil temperatures wheat is little affected, but at higher temperatures (25° C.) it becomes badly blighted. Maize, on the other hand, is chiefly affected at low soil temperatures (8° C.). This divergence in attack is correlated with the differing growth responses of the hosts at these temperatures. Growth of wheat seedlings at high temperatures and of maize seedlings at low temperatures results in the formation of thin cell walls which are easily penetrated by the mycelium. The same authors[15] have shown that both wheat and maize seedlings, grown in soil the moisture content of which is reduced to 30 per cent. of its water-holding capacity, are badly blighted at all soil temperatures. Wheat seedlings, grown at a suitable temperature but under weak illumination, are severely attacked, whereas with normal illumination they remain healthy.

Crop rotation usually reduces the incidence of this disease, although it is particularly dangerous to grow wheat after maize. Where the disease is prevalent, special care should be taken in the selection of seed grain.

There is urgent need for further investigation of the seedling blights and foot-rots of cereals which are associated with fungi having a Fusarium stage in their life-cycle (see also pp. 349, 350).

Sphaerostilbe, Tulasne

Perithecia as in *Nectria.* Conidial fructifications stalked, with a globose head on which the conidia are formed.

Sphaerostilbe repens, Berk. and Br.

Perithecia dark red, about 0·5 mm. in diameter; ascospores 2-celled, pale brown, $19–21 \times 8\,\mu$.

Conidial fructifications 2–8 mm. high, 0·5–1 mm. in diameter; stalk pink at first, then red-brown, tomentose; head white, globose; conidia variable in shape, unicellular, hyaline, $9–22 \times 6–10\,\mu$.

Mycelium forming red-brown or blackish, flattened rhizomorphs, 2 mm.–1 cm. broad, 0·5–2 mm. thick; rhizomorphs marked by a median groove and short, oblique lateral processes.

A root disease of plantation rubber in the eastern tropics caused by this fungus has been described by Petch[16] and Brooks[17]. It occasionally affects the underground parts of other tropical plants, e.g. tea and arrowroot, and Nowell[18] states that the same or a closely related species is responsible for the ' red root ' disease of limes in Dominica.

S. repens begins life as a saprophyte, often on dead stumps, whence it passes by means of rhizomorphs to the roots of rubber trees, either directly or through the soil. The fungus passes along the larger roots to the collar of the tree, whence the whole root system may be invaded. The branches die back slowly, and ultimately the tree is killed. In the host plant the fungus spreads chiefly along the line of the cambium by means of reddish-brown rhizomorphs, the remains of which can be seen on the wood long after the root is dead. The conidial fructifications are first produced on dead tissues near soil level, and are followed by perithecia.

In Malaya the disease is practically confined to old rubber trees growing in poorly drained, low-lying land. Trees which are dying back through attack by *S. repens* should be grubbed, and any stumps close by should be removed in order to prevent a possible extension of the disease. The affected area should be isolated by means of a trench to prevent the rhizomorphs from spreading through the soil.

S. musarum, Ashby, is a closely related species which attacks the root-stocks of bananas in Jamaica.

Sphaerostilbe flavida, Massee *

This fungus causes a serious disease of coffee in the Western Hemisphere, forming scattered, circular, whitish spots on the leaves, which soon turn yellow and fall. Similar spots occur on the young stems and berries. The 'conidial' stage, formerly known as *Stilbum flavidum*, Cooke, consists of yellowish pin-like structures. The heads of these 'fructifications' do not form spores, but become detached and serve as reproductive organs. The disease is most destructive where the shade is excessive.

Polystigma, De Candolle

Perithecia formed in a stroma embedded in a leaf; asci 8-spored; ascospores unicellular, hyaline.

Polystigma rubrum, (Pers.) DC.

In Britain this fungus affects the leaves of the Blackthorn and Bullace (*Prunus spinosa* and *P. insititia*), especially in localities near the sea; in Central Europe it occurs also on domestic varieties of plums. The fungus produces large red stromata in the leaves, on which spermogonia, with curved, supposedly functionless spermatia are formed during the summer. After leaf-fall the stromata become black, and perithecia are formed in them. The ascospores are ejected in the spring at about the time the new foliage is appearing.

Epichloe, (Fr.) Tulasne

Stroma effused, sheathing the host, at first white and abstricting conidia, becoming bright orange. Perithecia immersed; asci cylindric, 8-spored; ascospores filiform, many-celled, hyaline.

Epichloe typhina, (Fr.) Tul. Choke.

The stromata of this fungus form sheaths 2–5 cm. long around the basal parts of the leaves and stems of grasses, especially cocksfoot (*Dactylis glomerata*). The stromata are at first white and then golden yellow or orange in colour. Affected plants rarely flower because much of the mycelium fills the spaces between the leaves of the fertile shoot and prevents the panicle from protruding. Sampson ([18a]) states

* Recent work indicates that this fungus has been wrongly named. Maublanc and Rangel have shown that the perfect stage is an Agaric, *Omphalia flavida* (*Bull. Soc. Myc. d. France*, 1914). This has been confirmed by Ashby (*Kew Bull.*, 1925).

that the mycelium is perennial and is perpetuated by vegetative propagation of the host. In *Festuca rubra* she points out that the mycelium also occurs in the seeds of affected plants, particularly outside the aleurone layer and between the endosperm and the scutellum, so that the fungus persists from generation to generation.
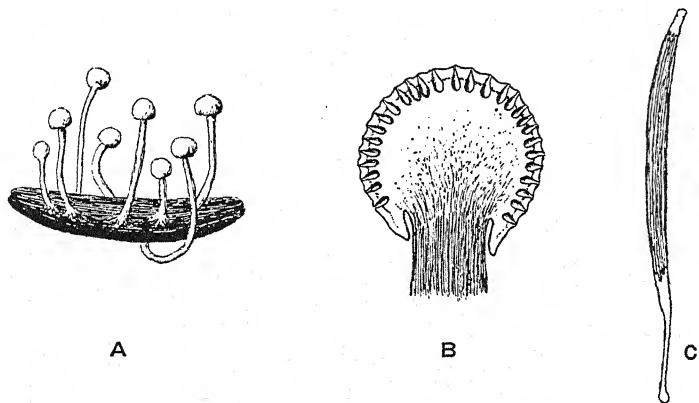
FIG. 31. *Claviceps purpurea*, (A) germinating sclerotium, slightly magnified; (B) section of stroma with perithecia, × 8; (C) ascus with spores, × 250. (*R. W. Marsh.*)

Claviceps, Tulasne

Stroma erect with a long sterile stalk and a globose head, arising from a sclerotium. Perithecia sunk in the head of the stroma; asci cylindrical, 8-spored; ascospores filiform, unicellular, hyaline.

Conidia formed at the extremity of the young sclerotium, oval, unicellular, hyaline.

Claviceps purpurea, (Fr.) Tul. Ergot.

Sclerotium purplish-black or black, elongate, 10–20 × 2–3 mm. Stroma pinkish or faintly purple, stalked. Perithecia with very slightly protruding ostioles; ascospores filiform, unicellular, hyaline, 60–70 × 2 μ.

Conidia small, oval, unicellular, hyaline.

The ergot fungus transforms the ovaries of cereals and grasses into hard, black sclerotia, which protrude from the ears. Among cereals it is most common on rye. It is rare on wheat in England, except occasionally on 'Rivet' and on hybrids between this and other varieties. Ergot frequently occurs on grasses, including perennial and Italian rye grasses.

Infection of the flowers takes place by means of ascospores. The mycelium passes into the ovary, which becomes transformed into a dense mass of hyphae. The upper part of the young sclerotium secretes a sugary fluid and abstracts numerous conidia (the Sphacelia or honey-dew stage), which are disseminated by insects or wind to other flowers, causing infection. The sclerotium grows considerably in length and ultimately becomes hard and dark-coloured. Some of the ergots fall to the ground, or they may be sown with the grain. If the ergots are not buried too deeply, they germinate in the spring to form numerous faintly purple, stalked stromata, the heads of which appear just above soil level. The ascospores are liberated at about the time cereals and grasses come into flower.

Stäger [19] has shown that there are several biologic forms of this fungus, the form on *Lolium perenne* and *L. italicum* being incapable of infecting rye and other cereals.

Although the sclerotia are of medicinal value, they also contain poisonous principles which produce gangrene of the extremities and cause abortion. It is dangerous for ergots to be included with grain that is to be ground into flour for human consumption. The ergots are also harmful to animals, and ergotized Italian rye-grass should not be fed to lambing ewes because of the danger of abortion. In the English fenlands, where oats are sometimes grown after rye, self-sown rye plants in the oats are often ergoted although the previous rye crop was healthy. Care should be taken in feeding such oats to horses.

DOTHIDEALES

Perithecia embedded in a hard, black stroma, without clearly differentiated walls. Conidial stages occur.

Plowrightia, Saccardo *

Stroma black, formed within the host tissues, then erumpent; asci cylindric, 8-spored; ascospores uni-septate, hyaline.

* This genus is considered by Theissen and Sydow (20) to be identical with *Dothidella*, but as the stroma of *Plowrightia* is markedly erumpent at maturity, it seems best to retain this genus.

Plowrightia ribesia, (Pers.) Sacc. (= *Dothidella ribesia,* (Pers.)
Theiss. & Syd.)

Stroma black, oval, about 4×2–3 mm. Ascospores fusiform with
pointed ends, septum nearly median, hyaline, 18–21×5–$6\,\mu$.

In young cultures the mycelium produces conidia which bud
in a yeast-like manner, somewhat resembling *Dematium pullulans,*
as pointed out by Hoggan [21].
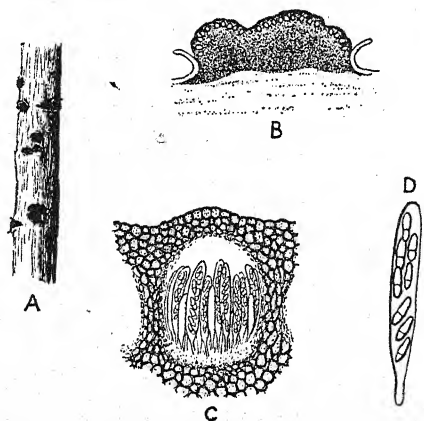
Fig. 32. *Plowrightia ribesia* on red currant : (A) general view, natural size ;
(B) section through stroma, × 15 ; (C) section through perithecium, × 100 ;
(D) ascus, × 200. (*R. W. Marsh.*)

This fungus causes a slow die-back of the branches of old
black and red currant bushes. Its parasitism has been investi-
gated by Hoggan [22]. The fungus is a wound parasite, entering
the bushes chiefly by way of snags left in pruning. The myce-
lium spreads downwards in the vessels of the wood and through
the bark and pith. Its progress in the pith may be impeded
by the formation of a cork barrier. As the branches die, black
stromata, containing perithecia, grow out through the bark.
The ascospores are violently ejected, one at a time, from the
asci in spring. On red currant bushes the fungus is often
associated with *Nectria cinnabarina. P. ribesia* has also
been recorded on gooseberry bushes.

Dothidella, Spegazzini

Stroma sunk in the host tissues, black. Asci 8-spored ; ascospores
uniseptate, hyaline.

Dothidella Trifolii, Bayliss-Elliott and Stansfield

Perithecia formed in over-wintered stromata; ascospores irregularly fusiform, 2-celled, hyaline, $24-26 \times 7-8 \, \mu$.

Pycnidia (= *Sphaeria Trifolii*, Pers.) formed in stromata; pycnospores catenulate, oval, hyaline, $5 \times 1\cdot5 \, \mu$.

Conidiophores (= *Polythrincium Trifolii*, Kunze) arising on the superficial stromata, tortuous; conidia obovate, 2-celled, brown, $20-22 \times 11-15 \, \mu$.

The perithecia of this fungus have been recently described by Bayliss-Elliott and Stansfield [23] and by Killian [24]. The latter refers it to the genus *Plowrightia*, but as the stroma is not erumpent it should be placed in the genus *Dothidella*. It has also been wrongly referred to the genus *Phyllachora*.

This fungus occurs on the leaves of *Trifolium repens*, *T. incarnatum*, and rarely on *T. pratense*, causing the formation of black spots on the under surface of the leaves. It is almost innocuous to *T. repens*, but has a serious effect on *T. incarnatum*. Conidiophores are formed on the almost superficial stromata which first appear on the leaves. Later in the season pycnidia are formed in other stromata, and during the winter perithecia are produced. The ascospores are liberated in the spring and serve as the first infecting units.

Phyllachora, Nitschke

Stroma sunk in the host tissues, usually black. Perithecia embedded in the stroma; asci 8-spored; ascospores unicellular, hyaline.

Phyllachora graminis, (Pers.) Fckl.

This fungus commonly produces black spots on the leaves of grasses, especially *Dactylis glomerata* and *Agrostis stolonifera*, but it is practically harmless. The black spots are stromata, in which perithecia are formed.

Other species of *Phyllachora* occur on the leaves of various kinds of plants.

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FUNGUS DISEASES *(continued)* : SPHAERIALES

SPHAERIALES

PERITHECIA dark-coloured, corky or carbonaceous in texture, sometimes embedded in a stroma. Pycnidia and other conidial stages occur.

Acanthostigma, de Not.

Perithecia free, spherical, black, with bristle-like appendages; ascospores spindle-shaped, 1–4 septate, hyaline.

Acanthostigma parasiticum, Sacc. (= *Trichosphaeria parasitica*, Hartig)

The mycelium grows on the under side of branches of the silver fir and spruce in Central Europe, from which it spreads to the lower surface of the leaves, fastening them firmly to the branches. Mycelial cushions are formed on the leaves. Haustoria penetrate the epidermal cells, and the leaves become brown and die. The minute, dark brown perithecia are formed in the mycelial cushions. The ascospores are usually 3-septate.

Herpotrichia, Fuckel

Perithecia free, black, embedded in a felt of long brown hairs; ascospores spindle-shaped, usually 4-septate, hyaline or light brown.

Herpotrichia nigra, Hartig

This fungus attacks the shoots of conifers growing at high elevations, and is particularly destructive in nurseries. The brown mycelium envelopes the leaves and twigs, matting them together and killing them. Most of the mycelium is external, but some hyphae penetrate the tissues. The perithecia are embedded in the superficial mycelium. The fungus grows well at low temperatures, and often spreads actively under snow.

Rosellinia, Ces. and de Not.

Perithecia free, usually surrounded by a mycelial felt, spherical, black, brittle ; ascospores elliptical or spindle-like, unicellular, brown to black.

Conidiophores of various types, sclerotia, and rhizomorphic strands occur. The hyphae often have pear-like swellings.

Rosellinia quercina, Hartig

Perithecia seated on a blackish mycelium, spherical with a minute papilla, about 1 mm. in diameter ; asci 8-spored ; ascospores fusoid with acute ends, unicellular, brown, 28×6-7μ.

Hartig[1] has described a serious root disease of young oak trees caused by this fungus. It has also been recorded on beech and sycamore in Denmark by Rostrup. The mycelium invades the rootlets, whence it passes into the main roots by means of fine hyphae, which arise from sclerotia-like bodies formed at the junctions of the rootlets with the large roots. The first symptom of attack is a yellowing of the foliage. The mycelium spreads back to the collar of the tree, when death ensues. The mycelium on the roots is white when young but brown when old. It frequently embraces the root in a dense covering of fine rhizomorphic strands. Black sclerotia about the size of a pin's head occur on the dead roots ; perithecia are formed later. Conidiophores with branches in whorls have also been described. The fungus grows through the soil by means of rhizomorphs, so that the disease often spreads centrifugally. The sclerotia may contaminate the soil for some years.

The disease is most prevalent in wet seasons. Hartig advises the isolation of diseased areas by means of trenches.

Rosellinia necatrix, (Hart.) Berl. White Root Rot.

A fungus, long known as *Dematophora necatrix*, commonly attacks the roots of vines, fruit trees (especially apple), and other plants. Viala[2] considers the fungus to be a Rosellinia, the perithecia of which occur only on roots that have been dead for some considerable time. Viala states that the perithecia have no ostiole.

The fungus spreads through the soil by rhizomorphs, which

are narrower and show less differentiation between rind and medulla than those of *Armillaria mellea* (p. 295). Small roots are first attacked, from which the fungus passes into the main roots. The whole of the root system is ultimately enveloped in a white mycelium, which becomes brown with age. The hyphae often show pear-shaped swellings. Black sclerotia, which occasionally give rise to conidiophores, are formed in the bark of the roots. In the early stages of attack the foliage becomes yellow and falls prematurely, then the branches die back, and finally the plant succumbs.

Nattrass [3] states that this disease is not uncommon on apple trees in the West of England. He has found that if the disease is seen at an early stage, the affected roots can be excised and the tree saved. Conidiophores (of the Graphium type) are somewhat infrequently seen in nature, but they develop readily in cultures. *R. necatrix* has recently been found to be destructive to Narcissus and Iris bulbs as well as to Arum corms and potato tubers in the Scilly Isles; it also occurs on elm roots there. Pethybridge * has reported that infection of potato tubers occurs through the lenticels by means of mycelial strands, the first symptom of disease being the formation of depressed, rotten areas around these organs. The mycelium subsequently grows deeply into the tuber.

White Root Rot is most troublesome in wet seasons and in badly drained soils, and appears to be favoured by somewhat high soil temperatures.

Rosellinia aquila, de Not.

On the Continent this fungus sometimes causes serious injury to mulberry trees, and in Scotland it has been recorded by Wilson [4] as killing spruce seedlings. Its mode of parasitism is similar to that of the two previous species, but the mycelium is less profusely developed. It is often a harmless saprophyte.

Rosellinia pepo, Pat.

This species causes a destructive root disease of cocoa trees and other cultivated plants in the West Indies. The mycelium,

* Communication made to the British Mycological Society, September 1926.

at first grey and then black, forms an irregular coat over the roots, and forms white, fan-like growths along the line of the cambium. The superficial mycelium may extend up the stem for several inches. Where the soil is rich in decaying matter the rhizomorphs spread readily through the surface layers.

Conidiophores of the Graphium type are formed in great abundance on the dead tissues and also on plant débris in the soil; the conidia are oval and unicellular. The perithecia, which are nearly smooth, are rarely found.

The disease often occurs in plantations established on old forest areas; it may spring from trees grown for shade, or it may arise directly in wet, densely shaded soils of high humus content. When a large part of the root system is affected, a thinning and yellowing of the foliage is seen; the tree usually dies suddenly as soon as the collar is invested by the mycelium. The disease tends to spread centrifugally.

Nowell[5] points out that the disease is most prevalent in damp, sheltered plantations, so that all measures that promote free ventilation between the trees should be taken. Trees in an early stage of disease can sometimes be saved by exposing the root system and excising the affected parts. Badly diseased trees should be completely removed and burnt; the infested area of soil should be surrounded by a trench, preferably square in shape, and deep enough to sever all the roots.

Rosellinia bunodes, (Berk. and Br.) Sacc.

This species is closely allied to the preceding, and, like it, attacks the roots of tropical plants, including limes in the West Indies, coffee in Southern India, and tea in Java and Ceylon. The superficial mycelium consists of black strands which thicken into irregular knots at intervals. Both the bark and the wood are permeated by rhizomorphs, which appear as black lines when the wood is cut across. Conidia of the same type as those of the preceding species occur, but the perithecia, which are covered with minute warts, are formed much more abundantly than in *R. pepo*.

Control measures are essentially the same as for *R. pepo*. Petch[6] points out that in diseased tea areas the prunings

should not be buried, as these encourage the growth of the fungus, and that green manuring should be temporarily abandoned in infested soil. The isolation trenches should be periodically inspected and cleaned out.

Rosellinia arcuata, Petch

This species is more commonly the cause of root disease of tea in Ceylon than is *R. bunodes*. The superficial mycelium on dead roots consists of black, woolly strands which are closely interwoven. Within the tissues, white stars of mycelium are found between the bark and the wood. The mycelium may extend some distance up the stem, on which it is at first greyish and then black. Conidia, borne on short bristle-like stalks, form a whitish powder over the black mycelium. Perithecia, which rarely occur, are found in clusters on the superficial sheets of mycelium.

Petch[7] states that in tea plantations in Ceylon an attack often originates in accumulations of dead leaves. He considers that the conidia are the chief means of disseminating the disease, and urges that dead bushes should be set on fire *in situ* before being uprooted and burnt.

New Zealand White Root Rot

This disease was attributed by Massee[8] to *Rosellinia radiciperda*, but, according to Cunningham[9], there is no adequate proof of this. The disease, which is similar to that caused by *R. necatrix*, is known only in New Zealand, where it occurs on the roots of fruit trees planted on areas previously under forest or scrub. Affected trees show a yellowing of the foliage, accompanied by a dying back of some of the branches. Recovery never occurs. The superficial mycelium is not particularly conspicuous, but the hyphae show the pear-shaped swellings characteristic of many Rosellinias. Between the bark and the wood the mycelium forms a fine white layer. Cunningham[9] confirms the presence of sclerotia and conidiophores, the former contaminating the soil for an indefinite period. Infected trees should be destroyed.

Ceratostomella, Saccardo

Perithecia with long, hair-like necks; asci egg-shaped, 8-spored,
the ascus walls soon disintegrating; ascospores unicellular, hyaline.
Conidial stages of diverse types occur.

Ceratostomella Piceae, Münch

This is one of the species into which Münch [10] has split *C. pili-
fera,* (Fr.) Wint. It occurs commonly on the sap-wood of felled
spruce and other conifers, but, according to MacCallum [11], does
not stain the wood like other species of the genus, and pene-
trates only a short distance into the tissues. Conidiophores of
the Cladosporium and Graphium types occur in the life-cycle.
Perithecia are formed abundantly on the surface of the wood.

Ceratostomella Pini, Münch

This species is chiefly responsible for the 'blueing' of the sap-
wood of felled Scots pine, the discoloration being due to the
coloured hyphae and to substances deposited in the cells of the
wood. Staining of this kind reduces the value of the wood,
although the fungus does not seriously disintegrate the tissues.
In Scotland logs are sawn immediately after felling in order
to obviate 'blueing'. Standing trees attacked by bark beetles
are occasionally invaded by this fungus. The perithecia have
much shorter necks than those of *C. Piceae.*

Ceratostomella fimbriata, (E. and H.) Elliott

Formerly called *Sphaeronema fimbriatum,* this species causes
a serious disease of the tapped bark of the Para rubber tree
in some parts of Malaya, as described by South and Sharples [11a],
where the disease is known as Mouldy Rot. The first signs
of attack are sunken blotches in the bark just above the
tapping cut, which spread and coalesce to form a depressed
band. The diseased tissues darken and become covered with
a greyish mould through which small black bristles (the
necks of the perithecia) subsequently protrude. At a later
stage the bark rots completely and exposes the wood, which
is discoloured superficially. Continuous wet weather is essen-
tial for an epidemic of Mouldy Rot, but the disease usually

develops only where the bark is thin. In addition to asco-spores, hyaline 'endoconidia' and dark 'macrospores' are produced in the life-cycle of the fungus. When the disease appears, tapping should be suspended for two weeks, and the tapped bark of affected trees should be painted two or three times with 50 per cent. brunolinum. The tapped bark of healthy trees in the vicinity should be painted with 10 per cent. brunolinum, and the tapping knives should be sterilized before use.

Halsted and Fairchild[11b] have described a black rot of sweet potatoes which is caused by the same fungus in the United States.

Cucurbitaria, Gray

Perithecia gregarious on a stroma, spherical, glabrous, black; asci cylindrical, 8-spored; ascospores elliptical, muriform, brownish.
Pycnidia gregarious, spherical, black; pycnospores muriform, brownish.

Cucurbitaria Laburni, de Not.

This fungus is a wound parasite of laburnum trees, the branches being gradually killed. Dense groups of pycnidia and perithecia, which are sometimes mixed together, grow out through the dead bark.

Other species of Cucurbitaria are parasitic on the spruce (*C. Piceae*, Borthwick), silver fir (*C. pithyophila*, de Not.), and barberry (*C. Berberidis*, Gray).

Guignardia, Viala and Ravaz

Perithecia sunken, spherical or somewhat flattened, black; asci clavate, 8-spored; ascospores elliptical or fusiform, unicellular or unequally 2-celled, hyaline; paraphyses absent.
Pycnidia of the Phoma type commonly occur.

Guignardia Bidwellii, Viala and Ravaz   Black Rot of Grapes.

Perithecia minute, globose, subepidermal, erumpent; ascospores ovate to elliptical, unicellular, hyaline, 12–17×4–$5\,\mu$.
Pycnidia black; pycnospores ovate to elliptical, unicellular, hyaline, 8–10×7–$8\,\mu$. Spermogonia with filiform spermatia (microconidia) also occur.

This disease appears on the leaves, young stems and berries of the grape vine, and is very destructive both in the United

States and on the Continent.   On the leaves it forms circular brown spots. The berries are attacked when about two-thirds grown, the disease appearing on them as purplish or brown spots, which gradually spread. Ultimately the berries shrivel. Pycnidia occur in great abundance on the leaves and fruit, but perithecia are found only on overwintered berries.

Reddick [12] advises the removal of shrivelled berries that remain hanging on the vines, and the ploughing in of those on the soil. The vines should be sprayed with Bordeaux mixture when the buds begin to swell, again when the buds are expanding, and at intervals subsequently. If spraying late in the season is necessary, ammoniacal copper carbonate should be used instead of Bordeaux mixture in order to avoid spotting of the fruit.

Other species of Guignardia

G. Vaccinii, Shear, is the most important cause of 'scald' in cranberries, and *G. Camelliae* (Cooke), Butler, causes 'copper blight' of the leaves of the tea plant in Ceylon and Assam.

Mycosphaerella, Johans.

Perithecia sub-erumpent, membranous ; asci cylindrical to clavate, 8-spored ; ascospores elliptical, uni-septate, hyaline; paraphyses absent.

Conidial stages of diverse types occur.

The genus contains many parasitic species, but the life-histories of many are still obscure.

Mycosphaerella Fragariae, (Tul.) Lindau   Strawberry Leaf Spot.

Perithecia black ; asci few ; ascospores with acute ends, uni-septate, hyaline, 15×3–4μ.

Conidial stage = *Ramularia Tulasnei*, Sacc. ; conidiophores simple, arising from a rudimentary stroma ; conidia elliptical, 1–3 celled, hyaline, 20–40×3–5μ.

Strawberry leaves are often affected with reddish spots caused by this fungus. The centre of the spot gradually becomes greyish, but remains bounded by a reddish border. The tufts of conidiophores are whitish.   The stromata or sclerotial bodies from which the conidiophores arise may

survive the winter, and form fresh conidia in the spring. Peritheica are formed in the autumn, but rarely occur.
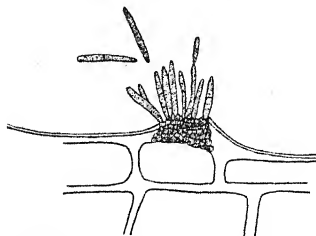
FIG. 33. *Mycosphaerella Fragariae*, section through a conidial pustule, × 315.
(*G. O. Searle.*)

In England the disease is seldom important, but, if serious, it may be checked by burning the beds superficially with the aid of straw in the autumn.

Mycosphaerella sentina, (Fr.) Schr.   Pear Leaf Fleck.

In the pycnidial stage (= *Septoria piricola*, Desm.) this fungus forms dry, brown spots on pear leaves, and may cause defoliation. Peritheica are only found on over-wintered leaves.

Mycosphaerella brassicicola, (Fr.) Lindau   Ring Spot of Brassicae.

This fungus occurs commonly on broccoli, cabbage, and Brussels sprouts in England, and may cause partial defoliation, but it often does no appreciable harm.   Brassicae of a soft habit of growth consequent on heavy nitrogenous manuring are the most liable to severe attack.   Brown spots are formed on the leaves, particularly the older ones, on which the pycnidia are arranged in concentric zones.   The spores, which measure $2 \cdot 5 – 4 \cdot 5 \times 1 \cdot 5 – 2 \cdot 5 \ \mu$, exude from the pycnidia in pink tendrils. Peritheica occur somewhat rarely in England.

Weimer [12a] has investigated the disease in California, where it causes serious damage to broccoli and cauliflower in wet seasons.

Mycosphaerella Ulmi, Kleb.

Klebahn [13] has found this to be the perithecial stage of the fungus formerly known as *Phleospora Ulmi*, Wallr., which causes a brown spotting of elm leaves.

Mycosphaerella laricina, Hartig   Larch Needle Cast.

The most serious needle disease of the larch is caused by this fungus, which has been investigated by Hartig [14].   The leaves are affected with brown spots, and fall rapidly.   Small black pustules appear on the spots and give rise to two types of conidia, the first being minute and ungerminable, the others being 30 μ long and ultimately 3-septate.

The disease is especially severe on larch interplanted with spruce, chiefly because the larch needles fall on the spruce branches, in consequence of which re-infection by ascospores is facilitated in the following year.   Larch woods at high elevations are comparatively unaffected.   The disease has been reduced by interplanting with beech after the larch has been thinned, as the young beech foliage acts as a screen impeding the ascospores from rising to infect the larch needles above.

Mycosphaerella citrullina, (C. O. Smith) Grossenbacher

This species attacks muskmelons, cucumbers, and other cucurbits in the United States and Japan.   In muskmelons the disease is most common as a spotting of the leaves and a wilt of the stems, but in cucumbers the fruit is most often affected.   Infection of the stem usually occurs at the leaf axils, but the fungus does not spread far up and down the stem.   The lesions in the stem are characteristically sunken.   The pycnidia and perithecia are black, the pycnospores being uniseptate.   The disease can be controlled by spraying with Bordeaux mixture.

Sphaerulina, Saccardo

Perithecia black ; asci 8-spored ; ascospores long, with several cross walls, hyaline ; paraphyses absent.
   Pycnidia also occur.

Sphaerulina rehmiana, Jaap

The pycnidial stage of a fungus, long known as *Septoria Rosae,* Desm., has been connected with *Sphaerulina rehmiana* by Klebahn [15].   The fungus causes a leaf scorch of roses, and may lead to serious defoliation of nursery stock.   The first sign of disease is the appearance of small yellowish patches on the leaves, which spread, turn brown, and finally may fall away, giving a ' shot-hole ' effect.   The pycnidia, which develop on
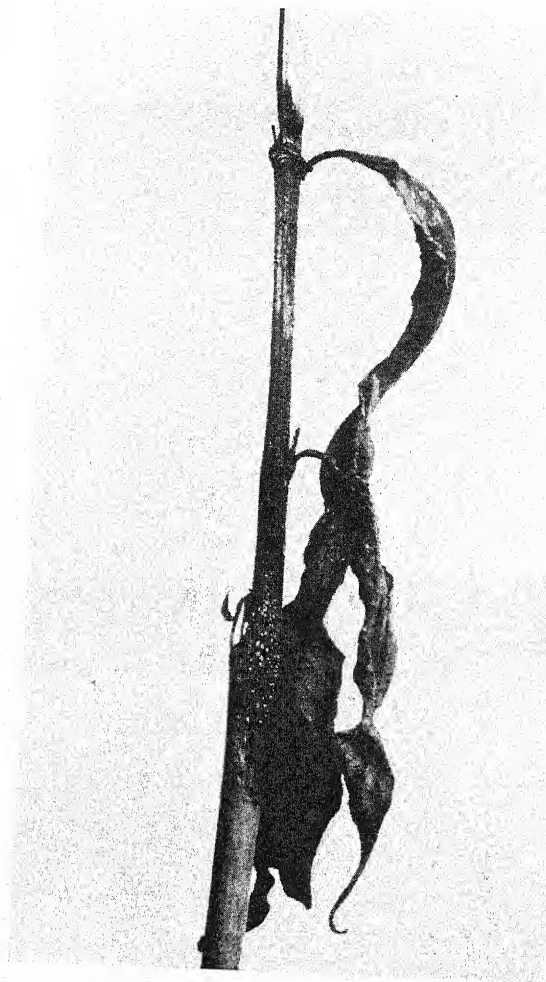
FIG. 34. *Physalospora miyabeana*, showing infection of stem of *Salix vitellina* from leaves after placing conidia near the base of the lamina ; acervuli visible on the stem.   Natural size.   (*R. M. Nattrass.*)

the brown spots, contain septate, thread-like spores. Perithecia are found only on overwintered leaves.

Physalospora, Niessl.

Perithecia sub-globose, black, with an erumpent ostiole; asci clavate; ascospores long, oval, unicellular, hyaline; paraphyses present.

Conidial stages of various kinds occur.

Physalospora miyabeana, Fukushi   Black Canker of Osier Willows.

Perithecia solitary or in small clusters, globose, dark brown, 100–170 μ in diameter; asci numerous, clavate; ascospores uniseriate or sub-distichous, oblong-ellipsoidal, unicellular, hyaline, 15–17 × 5·5–7 μ; paraphyses slender.

Acervuli (Gloeosporium stage) gregarious, light brick-red; conidia ellipsoidal, hyaline, 13–23 × 3·8–6·8 μ.

Fukushi[16] has described this fungus as causing a leaf spot and stem canker of *Salix purpurea* var. *angustifolia* in Japan. Nattrass[17] has investigated a similar disease of osier willows (especially *Salix pentandra*) in England, which is due to the same or a closely related fungus. Nattrass has found that after attacking the leaf, which turns black and droops, the fungus often grows down the petiole into the stem, where a canker is formed. It also attacks the ends of the young twigs, causing them to shrivel and bend over. Acervuli of the Gloeosporium type, and then perithecia, are formed on the stem lesions. The fungus is a true parasite, the germ tubes penetrating the cuticle.

Cankered rods are useless for basket-making and should be destroyed. Nattrass suggests that the osiers should be sprayed with Bordeaux mixture in the spring to prevent infection.

Nattrass[17] states that the exact systematic position of the fungus is uncertain, and that it may perhaps belong to the genus Glomerella.

Johnson[18] attributed a canker of osier willows to *P. gregaria,* Sacc. Schwarz[19] states that *P. Salicis,* Fuckel, is a weak parasite following lesions caused by *Fusicladium saliciperdum,* (All. & Tub.) Tub.

Physalospora Cydoniae, Arnaud (= *P. Malorum,* (Berk.) Shear)

Perithecia globose, with a short, papillate ostiole, black; asci clavate; ascospores ellipsoidal or inequilateral, unicellular, hyaline or greenish yellow, $23–34 \times 11–15\,\mu$.

Pycnidia (= *Sphaeropsis Malorum,* Pk.) globose, blackish, erumpent; pycnospores oblong-elliptical, brown, unicellular, $22–32 \times 10–14\,\mu$.

Only the pycnidial stage of this fungus has yet been found in England. The fungus is one of the causes of the appearance of brownish spots on the leaves of apple, pear, quince, and other trees, but spores are rarely formed on these spots. It produces cankers in young and old stems, and Cunningham [20] states that it is the common cause of apple and pear canker in New Zealand. The cankered areas are elliptical in shape and show numerous cracks arranged in concentric zones. Both pycnidia and perithecia occur on the cankers. The fruit also may be infected, the first sign of disease being small circular brown areas; these extend until the whole fruit becomes spongy, and ultimately black and mummified. The disease is known as 'black rot' of apples when it attacks the fruit.

In the eastern parts of N. America Hesler [21] states that this disease may be very serious, but in England it is usually of no importance. Bad cankers should be excised.

Venturia, Cesati & de Notaris

Perithecia more or less sunk in the substratum, ostiolate, membranous, dark coloured, with a few setae chiefly towards the top; asci sac-like, later becoming much elongated; ascospores elliptical, uniseptate, hyaline to olive green; paraphyses quickly becoming disorganized.

Conidial stages occur.

Venturia inaequalis, Aderh. Apple Scab (Black Spot).

Perithecia flask-shaped, $90–170\,\mu$ diameter, averaging $138\,\mu$, neck projecting more or less through the epidermis, with a few brown setae; asci at first oblong, $60–70 \times 6–11\,\mu$, with biseriate asco-spores, later, when ejecting spores, much elongated with the spores uniseriate, 8-spored; ascospores unequally 2-celled, upper cell smaller, $12–15 \times 6–7\,\mu$, pale olive-green.

Conidial stage = *Fusicladium dendriticum*, Fuckel ; conidiophores septate, brown, wavy, $50–60 \times 4–6\,\mu$; conidia terminal, obclavate, yellowish-olive, at length becoming uniseptate, $30 \times 7–9\,\mu$.
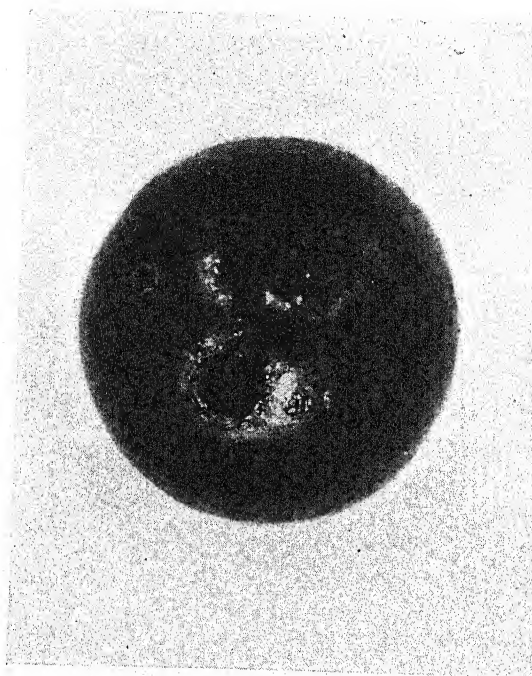
FIG. 35. *Venturia inaequalis*, scab spots on apple. Natural size.

The fungus occurs on the young twigs, leaves and fruits of apple trees and other species of Pyrus except the pear. The disease caused by it is one of the most serious that fruitgrowers have to contend with. The spores first infect the expanding leaves and sepals. The details of infection have been worked out by Wiltshire[22]. The germ-tube forms an appressorium, from which a hypha penetrates the cuticle. The mycelium spreads laterally in the cuticle, forming cushions, which give rise to conidiophores that break away the upper part of the cuticle. With resistant varieties the cuticle is also penetrated, but the mycelium grows feebly and fails to form conidiophores. Rarely, leaves may be so badly attacked as to fall prematurely.

Conidia formed on the leaves or sepals infect the young fruits, on which circular, somewhat sunken, black spots with a whitish margin are produced.

In severe attacks the fruit may crack as it grows. Older fruits are not easily infected owing to better cuticular protection. Twigs of the current season are also affected, the fungus penetrating the bark and giving rise to conidial pustules in the following spring. These pustules are formed as swellings just under the surface of the twigs, and give the latter a characteristic appearance.

As the leaves die, the mycelium penetrates the tissues; perithecia, which liberate ascospores from March onwards, are formed in overwintered leaves lying on the ground. In England perithecia have only recently been found by Salmon and Ware [23], but they are now known to occur commonly. Under moist conditions ascospore-discharge may continue for several weeks. The new foliage may be infected either by conidia or by ascospores.

The scabbing or spotting of the fruit is the phase of the disease of greatest concern to the fruitgrower, as this reduces the market value considerably and also renders the stored fruit more susceptible to attack by other fungi. Some varieties are more susceptible to Scab on the twigs than on the fruit. Although varieties show considerable differences in susceptibility in an average season, these differences are largely obliterated in a year that is bad for Scab. The intensity of the disease varies much from season to season according to the weather, frequent rain in the early summer favouring it greatly. Soil conditions also play some part in determining the severity of attack, twig infection being usually commonest on heavy soils. In an average season the following varieties are most susceptible under English conditions: 'Worcester Pearmain', 'Lord Suffield', 'Bismarck', 'Cox's Orange Pippin'. In a bad season even 'Bramley's Seedling', 'Newton Wonder', and 'Lane's Prince Albert' may be severely affected.

The disease can be controlled in great measure by spraying with Bordeaux mixture or with lime-sulphur of specific gravity 1·01, or, with the more sensitive varieties (e. g. 'Cox's Orange

Pippin '), 1·003.   The varieties 'Stirling Castle' and 'Lane's Prince Albert' should not be sprayed with lime-sulphur as they are very susceptible to injury by this fluid.   At least two, and preferably three, applications should be given.   The first spraying should be done when the blossom buds are showing pink, the second just after the fall of the petals, and the third about three weeks later.   For the second and third sprayings it is advisable to use lime-sulphur of spec. grav. 1·005 or 1·003.   On the whole it is safer to use Bordeaux mixture for the post-blossom spraying as there is some danger of fruit-drop with lime-sulphur, especially if the latter was not used for the pre-blossom spraying.   The object of spraying is to prevent the establishment of the fungus on the leaves, whence the fruit is chiefly infected.   Keitt and Jones [24], who have recently studied the epidemiology of apple Scab in Wisconsin, call attention to the danger of early infection of the sepals, and advise spraying immediately after the fruit buds have begun to open.   Badly scabbed twigs should be cut out as far as possible.   The fallen leaves should be collected and burnt in grass orchards, and they should be dug into the soil during winter in plantations kept under clean cultivation.

Venturia pirina, Aderhold   Pear Scab.

This species differs only slightly from the preceding, chiefly in that the upper cell of the ascospores is longer than the lower.
Conidial stage = *Fusicladium pirinum*, (Lib.) Fckl.

The life-history of the fungus causing pear Scab is essentially the same as that of *Venturia inaequalis*, but the fungus can only infect the pear.   *Venturia pirina* grows on the under side of the leaves as well as on the upper surface, and it frequently causes a more severe cracking of the fruit than does the apple scab fungus.   Infection of the young fruit often occurs before that of the leaves.   Pears grown under shelter are rarely affected by the disease, probably because insufficient water is deposited on the leaves and fruits to allow of the initiation of infection.   In France paper bags are sometimes tied over young fruits of the best dessert varieties in order to prevent Scab attack.   The following varieties are most

susceptible to Scab on the twigs : 'Doyenné du Comice', 'Marie Louise', 'Pitmaston Duchess', 'Beurré Bosc', and 'Fertility'. The fruits of the last-named variety are often badly attacked by Scab.

The disease can be controlled by the measures applicable to apple Scab. Bordeaux mixture should be used for spraying, as lime-sulphur injures the foliage of some varieties of pears. The first spraying should be carried out immediately the petals have fallen, and the second spraying about three weeks later.

Venturia Cerasi, Aderh.

This fungus occasionally causes a scab of cherry fruits, but in England it has as yet been found only in the conidial stage, *Fusicladium Cerasi* (see p. 336). The perithecial stage occurs on the Continent.

Didymella, Saccardo

Perithecia devoid of hairs and ascospores hyaline, otherwise as in Venturia.

Didymella Lycopersici, Klebahn

Perithecia sub-globose, dark-brown ; asci cylindrical, 70–95×9–10μ, 8-spored ; ascospores spindle-shaped, uniseptate, hyaline, 16–$18 \times 5 \cdot 5$–$6 \cdot 5 \mu$.

Pycnidial stage $=$ *Diplodina Lycopersici*, (Cooke) Hollós. Pycnidia brown to brownish-black, 100–270μ diameter ; pycnospores uni-cellular to uniseptate, hyaline, $4 \cdot 5$–$17 \times 2 \cdot 5$–5μ.

This fungus causes a stem and a fruit rot of tomatoes, and a stem rot of Capsicums. It has been investigated by Brooks and Searle[25] and by Klebahn[26], who has found the perithecial stage in Germany. In Britain the fungus was formerly erroneously referred to *Mycosphaerella citrullina*.

In England the fungus causes a rot of the basal parts of the stems of tomato plants grown under glass, and, more rarely, of outdoor plants. Serious epidemics of this disease may occur in tomato cultivation under glass. The spores can infect uninjured tissues, and when the stem just above soil level is attacked the plant falls over and dies. The fungus also causes a rot of the fruits as they ripen. Pycnidia are produced on the diseased tissues, there being great variability

in the number of uniseptate spores. Klebahn[26] found the perithecia on over-wintered stems, but these have not yet been seen in England.

Didymella applanata, (Niessl.) Sacc., causes a spur blight of raspberries in Britain, on the Continent, and in N. America. The disease has been investigated by Osterwalder[27] and others. In N. America the fungus is known under the name of *Mycosphaerella rubina*.

Infection occurs at the nodes; the fruiting buds are frequently killed and the fungus spreads down the canes. There is a Phoma stage in the life-cycle, as described by Newall[28]; the perithecia are not mature until the summer following infection.

Didymellina, von Höhnel

Perithecia as in Didymella, but without paraphyses.

Didymellina macrospora, Klebahn   Iris Leaf Spot.

Perithecia globose, blackish; asci obclavate, 8-spored; ascospores oval-elliptical, slightly unequally 2-celled, light greenish-yellow, $31-54 \times 11-16 \mu$.

Conidial stage = *Heterosporium gracile*, Saccardo. Conidiophores olivaceous, somewhat zigzag in shape; conidia echinulate, 2–3 septate, olivaceous, $40-60 \times 18-19 \mu$.

This fungus causes a spotting of the leaves of *Iris germanica*, *I. florentina*, and *I. variegata*. The disease is first evident about midsummer as small brown spots surrounded by a water-soaked margin. The spots extend and coalesce, and entire leaves may become brown and shrivelled. The fungus may spread epidemically in wet weather and destroy most of the foliage. Infection occurs through the stomata, and the conidiophores subsequently protrude through these. Tisdale[29] in the United States found perithecia on over-wintered leaves, and identified them as *D. Iridis*, (Desm.) v. H., but Klebahn[30] states that this species has much smaller spores and is not associated with *Heterosporium gracile*.

The disease can be reduced by burning the affected leaves in the autumn.

Leptosphaeria, Cesati and de Notaris

Perithecia spherical, dark-coloured, with a papillate ostiole; asci club-like at first, but greatly elongating at the time of spore

discharge, 8-spored; ascospores elliptical to spindle-shaped, twice or more septate; yellow to dark brown; paraphyses present. Diverse conidial forms occur.

Leptosphaeria Tritici, (Gar.) Pass.

The fungus affects cereals on the Continent, and is perhaps commonest on wheat. The leaves and sheaths are attacked, the perithecia appearing as brown or blackish specks. The disease sometimes reduces the yield considerably, as the ears ripen prematurely and the grain does not fill well. The ascospores are 3-septate.

Leptosphaeria herpotricoides, de Not.

This species attacks the stems of wheat and rye in France, Germany, and Sweden, often causing the nearly ripe straw to break at the bottom. The perithecia appear as black specks on the lowest internode, the ascospores being 6-8 septate. According to Gaudineau and Guyot [31] the disease is more serious on winter than on spring wheat, and is most prevalent after a mild winter. They state that the disease is greatly reduced by spraying the fields with a 15 per cent. solution of sulphuric acid about the beginning of April.

Leptosphaeria coniothyrium, (Fuckel) Sacc.

Perithecia gregarious, spherical, black, with an erumpent ostiole; asci cylindrical, 8-spored; ascospores uniseriate, oblong, 3-septate, brownish, $10-15 \times 3-4 \mu$.

Pycnidial stage = *Coniothyrium Fuckelii*, Sacc.: pycnospores elliptical, unicellular, brownish, $2.5-5 \times 2-3.5 \mu$.

This fungus affects the stems of a considerable number of plants; it causes a wilt of raspberry canes, which is serious in the United States. Cunningham [32] states that it attacks the blackberry in New Zealand. Brownish-black pycnidia develop on the dead tissues, and perithecia commonly occur on diseased raspberry canes in the United States.

Ophiobolus, Riess

Perithecia scattered, sunk in the substratum, spherical, dark, with a somewhat elongated neck; asci cylindrical, 8-spored; ascospores thread-like, arranged parallel to each other in the ascus, with several cross walls, yellowish or hyaline; paraphyses present.

Ophiobolus graminis, Sacc. Whiteheads or Take All of Cereals.

Perithecia black, with a conical curved beak; ascospores needle-shaped, slightly curved, 3–5 septate, hyaline, 70–75 × 3–5 μ.

'Take-all' is a serious disease of wheat in Australia and the United States, and it sometimes causes considerable damage in European countries. The fungus attacks other cereals and wild grasses also, including couch (*Agropyrum repens*). The two popular names for this disease denote different phases of attack. 'Take-all' refers to the killing outright of groups of young plants, and 'Whiteheads' relates to a later or less severe attack which results in empty, bleached ears.

Infection takes place at about soil level through the leaf sheaths, culm or roots. The whole of the root system may be destroyed. The mycelium penetrates the vascular cylinder of the stem, and it often forms a brown felt on the outside of the stem and inside the sheaths. If the young plants are not killed or if attack occurs later, the lower part of the straw becomes brown and brittle and the ears are bleached in consequence of the cutting off of the water supply. The perithecia are embedded in the mycelial weft surrounding the base of the stem; they may also be formed in the lower leaf sheaths. Davis[33] states that the ascospores and probably the mycelium overwinter on stubble left in the ground.

Some varieties of wheat, e.g. 'Marquis' in N. America, are particularly susceptible to attack. The disease is most prevalent where there is no extended crop rotation and in wet, warm seasons. Weed grasses such as couch should be kept down, as these harbour the fungus. At harvest the straw should be cut so as to avoid the inclusion of the parts that contain the spores of the fungus; otherwise, the fungus may be disseminated with dung.

Ophiobolus herpotrichus, Sacc.

This species, which has been recorded in Italy and other countries, produces the same effect on wheat as *O. graminis*. The ascospores are much longer than in the latter, being 135–150 μ long.

Pleospora, Rabenhorst

Perithecia spherical, more or less erumpent at maturity, black; asci club-like or cylindrical, 8-spored; ascospores ovate, muriform, yellowish-brown or hyaline; paraphyses present.

Conidial stages of diverse types occur, including pycnidia. According to Brefeld [34] *P. vulgaris* has an Alternaria stage, and *P. herbarum* is the perithecial form of *Macrosporium commune.* Most species of Pleospora are saprophytic.

Pleospora pomorum, Horne

This is one of the many fungi which cause spotting of apples both on the tree and in storage. A general rot may also be induced by it. The disease has been described by Horne [35] and by Kidd and Beaumont [36]. The spots, which are brown in colour and 1–2 mm. across, arise at the lenticels, but in storage the fungus may spread extensively and cause a brownish black rot. Invasion occurs through the lenticels because these are largely unprotected by cuticle and cork, and because nutritive substances, which facilitate spore germination, exosmose into them as the fruit matures. Perithecia are formed in the diseased tissues, but these appear to be invariably sterile on the fruit, although they readily form spores in culture. A Stemphylium conidial stage also occurs in culture.

Pyrenophora, Fries

Perithecia as in Pleospora, but the upper part of the wall is provided with stiff hairs.

The conidial stages are chiefly Helminthosporium forms.

Pyrenophora graminea, (Died.) Drechsler (= *Pleospora graminea*, Died.) Leaf Stripe of Barley.

The perithecia described by Diedicke [37] as belonging to *Helminthosporium gramineum* are believed by both Drechsler [38] and Smith [39] to have belonged to *H. teres.* Smith, however, has found immature perithecia indubitably associated with *H. gramineum*, which differ only slightly from those of *H. teres.* The perithecia are formed in sclerotial bodies.

Conidial stage = *H. gramineum*, Erikss.; conidiophores short, in clusters of 2–6, sub-flexuose, light brown; conidia solitary, elongate-cylindrical, 1–7 septate, brown, $50–125 \times 14–21 \mu.$

This fungus occurs wherever barley is grown and is responsible for considerable crop losses. The fungus causes diverse symptoms according to the measure of its attack on the plant. Seedlings may be killed outright, but, more frequently, the first clear symptoms are the appearance of long brown stripes on the leaves, followed often by partial or complete failure of the ears to emerge; the grain may fail to develop ('blindness'*) or may become discoloured. Secondary infections of the leaves occur, but in these the brown areas may be of irregular shape, simulating attack by *H. teres*. Although secondary infections of the leaves are of little direct importance, they may be one of the chief sources of conidia which contaminate the grain.

FIG. 36. Conidia of *Helminthosporium gramineum*, × 175. (*R. W. Marsh.*)

The disease is transmitted by the grain, through attached conidia, by mycelium in the chaff, or by mycelium which has penetrated more deeply into the tissues. Upon germination of the grain the conidia also germinate or the mycelium re-awakens to activity. In this way the developing shoot is attacked, the inner surface of the coleoptile being usually the first part to be infected. The seedling may be killed, especially if the mycelium was in the embryo and endosperm, or it may be only slightly invaded by the mycelium. Ravn[40], who investigated the disease in Denmark, considered that the fungus often behaved in much the same way as a Smut fungus, keeping pace with the stem apex and passing out laterally to the leaves. Smith[41], however, has shown that the mode of parasitism, when the seedling is not killed outright, is unlike that of a smut fungus, but is of a peculiar kind in that the first leaf is infected through contact with the diseased inner surface of the coleoptile, the second leaf through contact with the enclosing sheath of the first leaf, and so on. This mode of infection, together with the tendency of the mycelium to

* 'Blindness' in barley is not always due to this fungus, but to other, at present unknown, causes.

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spread longitudinally between the veins, accounts for the appearance of long brown stripes on the leaves characteristic of the disease. The ears are also infected before emergence, through contact with diseased leaf sheaths outside them. The mycelium may enter the nodes of the stem slightly, but causes little harm in these hardened tissues. Occasionally the hyphae invade the delicate growing point of the stem and kill it. If the primary attack on the leaves is severe, the ear fails to emerge, or only partly emerges, and may fail to set grain. The ears which develop more or less normally, but which are slightly infected through contact with striped leaves, are a frequent source of contaminated grain, for in such ears the grain is not sufficiently attacked to ensure rejection.

The mycelium in the leaf is intercellular. Conidia are formed on the discoloured parts of the leaves and ears. In the autumn hard sclerotial bodies are formed in the old leaves, which produce perithecia in the following spring. Mature ascospores, apart from Diedicke's doubtful record, have not yet been seen.

Six-rowed barleys are generally more susceptible than two-rowed varieties, and winter-sown grain is more liable to attack than barley sown in spring. In certain seasons most English varieties may be severely affected. Attempts are being made to breed commercial varieties which will be resistant to attack. Efforts have been made to control the disease by disinfecting the grain with formalin and other substances (as described on p. 214), often with considerable success, but when the mycelium is within the grain it cannot be reached by the antiseptic. Grain harvested from fields affected by Leaf Stripe should not be used as seed.

Pyrenophora teres, (Died.) Drechsler (= *Pleospora teres*, Died.) Net Blotch of Barley.

The perithecia of this fungus have been described by Drechsler[38] and others in the United States, and by Smith[39] in England. The perithecia of a Helminthosporium described by Diedicke[37] probably belonged to this species. Diedicke originally gave the name *Pleospora teres* to a hypothetical perithecial stage of *H. teres*.

Perithecia brownish-black, 0·5 mm.; asci sub-cylindrical, 220–

230×30–$36\,\mu$, 8-spored; ascospores biseriate, 3-septate, light brown, 50–60 $\times$ 18–22 μ.

Conidial stage = *Helminthosporium teres*, Sacc. ; conidiophores short, in clusters of 2-4, sub-flexuose, brown ; conidia solitary, elongate-cylindrical, 1–10 septate, greenish-fuliginous, 30–175 $\times$ 15–22 μ.

' Net-blotch ' disease of barley, although common, does not affect the plant so seriously as does ' Leaf Stripe '. The fungus is carried over with the grain, and infection of the first leaf may arise as in *P. graminea*, the result being the formation of a pale stripe, which may become brown. The later leaves, however, rarely become infected by contact, so that the stripe form of the disease is rarely seen after the seedling stage. The ears remain free from primary attack, but the grain often becomes contaminated through the chaffs being secondarily infected. Conidia formed on the first leaves bring about secondary infections of the foliage, which appear as irregular, brown blotches, somewhat net-like in appearance. The secondary infections may assume epidemic proportions, but they rarely reduce the yield seriously.

Sclerotial bodies are formed on the dead leaves during the autumn, and perithecia develop in these in the spring, the ascospores causing infections of the secondary type.

Pyrenophora Avenae (?) (= *Pleospora Avenae*, Died.) Leaf Spot of Oats.

Diedicke [37] suggested *Plcospora Avenae* as a name for the hypothetical perithecial stage of *Helminthosporium Avenae*, (Bri. & Car.) Eid. The conidiophores and conidia are practically indistinguishable from those of *H. teres*.

The life-history is essentially the same as that of *P. teres*. The fungus is carried over with the grain, and the disease may kill the seedlings outright. Alternatively, the first leaf may show a stripe similar to that caused by other species of Helminthosporium, but plants affected in this way often grow away from the disease. Secondary infections, forming brown spots, commonly affect the early foliage, but the later leaves are often free from attack. The ears are affected in a secondary manner, if at all.

Pycnidia sometimes occur on the glumes, pales and kernels of diseased grain. Perithecia of the Pyrenophora or Pleospora type have also been found on seed grain, but it is not yet known whether these are related to *H. Avenae*.

Oats for seed purposes should be selected from fields which were free from the disease.

Other species of Helminthosporium

Although perithecia have not yet been associated with the following species, it is convenient to describe them here as they are closely related to other species of Helminthosporium which attack cereals.

Helminthosporium sativum, Pammel, King and Bakke

Conidiophores solitary or in clusters of 2–3, fuscous, septate; conidia solitary, often slightly curved, 6–11 septate, dark olivaceous, with a thick peripheral wall, 60–130×15–30μ.

This species attacks wheat, barley, rye, and certain wild grasses, causing a seedling blight, root rot, foot rot, leaf spot, and discoloration of the grain. It is one of the most serious fungus diseases of cereals in the United States and Australia; it has recently been found occasionally in England by Smith[41], causing a foot rot of barley. The fungus has been studied in the United States by Stevens[42], Christensen[43], Dosdall[44], and Drechsler[38].

The first symptom of disease in a field is the damping-off of some of the young plants. Others become dwarfed and discoloured at the base of the stem and first leaf. Such plants may gradually succumb or recover, and they frequently tiller excessively, although partial attack may also prevent tillering. The roots of such plants are more or less permeated by the parasite, and, even if these plants mature, the heads fill poorly and the grain is often shrivelled owing to malnutrition.

Conidia are formed on the diseased parts, and these infect the older leaves secondarily, causing small brown spots, and also the glumes and grain, which may be discoloured or completely destroyed. A black discoloration of the embryo in the grain known as 'black point' is often caused by this fungus, as pointed out by Henry[45]. The fungus is carried

over on, or in, the grain, and it may persist in the soil. It grows best at fairly high temperatures.

Christensen [46] has described many forms of the fungus, which differ greatly in pathogenicity and cultural behaviour, but strains of the fungus from wheat infect barley, and reciprocally. 'Marquis' and Red Durum wheats are very susceptible to root infection, but 'Kanred' is resistant. The disease is difficult to control. Proper rotation and the use of clean seed reduce the disease, but do not always eliminate it. Further search for resistant varieties offers the best promise of control.

Helminthosporium turcicum, Pass.   Leaf Blight of Maize.

This fungus occurs commonly on maize in Italy, India, South Africa, and the United States. It produces round yellowish spots on the leaves, which extend longitudinally until much of the lamina is affected. The diseased areas gradually darken owing to the formation of conidiophores. Ultimately the attacked leaves dry up and appear as if burnt. The disease is sometimes so severe as to prevent the grain from ripening.

Helminthosporium Oryzae, B. de H.   Brown Spot of Rice.

This species, which causes much damage in Japan, forms elongated brown spots on rice leaves. The spikelets also are affected. The disease is seed-borne, but it can be prevented by immersing the grain before sowing in water at a temperature of 52° C. for 10 minutes.

Gnomonia, Cesati and de Notaris

Perithecia with a more or less elongated neck ; asci elliptical or fusoid, with a thickened apex transversed by a ' canal ' ; ascospores elongate, 2- or 4-celled, hyaline ; paraphyses absent.
   Conidial and spermatial stages occur.

Gnomonia erythrostoma, (Pers.) Auersw.   Cherry Leaf Scorch.

Perithecia with a long protruding neck ; ascospores unequally 2-celled, the small lower cell being ungerminable. Slender, curved spermatia, which have not been seen to germinate, are formed in spermogonia.

The popular name is given to this disease because leaves in the later stages of attack appear as if scorched. Affected leaves hang on the trees throughout the winter. The disease occurs in most districts where sweet cherries are grown considerably; it is rarely of importance, although the fruit may be reduced in quantity and be malformed where trees have been attacked for several years. The young leaves are infected by ascospores shot forth from perithecia which have matured in the over-wintered leaves. The first sign of attack is the development of large yellowish patches which spread and turn brown. The mycelium is intercellular, but Brooks[47] states that haustoria are not formed. Spermogonia occur on the under surface of the leaves during summer. The mycelium passes down the petiole and prevents the formation of an absciss layer, so that the leaves are not shed.

The disease can be prevented by stripping off the leaves which hang on the trees during the winter, thereby destroying the only source of infection. Spraying the trees with Bordeaux mixture when the leaves are unfolding is recommended by some authorities as a means of preventing re-infection. In England the varieties 'Florence' and 'Waterloo' are very susceptible, but 'Black Heart' is only slightly attacked, and 'Turk' and 'Crown' are practically immune.

Gnomonia veneta, (Sacc. and Speg.) Kleb.   Plane Tree Scorch.

Perithecia with a long protruding neck; asci clavate, generally bent at right angles at the base; ascospores unequally 2-celled, the upper cell longer, $14–19 \times 4–5\,\mu$.

Several conidial stages occur, the commonest being that formerly known as *Gloeosporium nervisequum*, in which the acervuli are subcuticular and form unicellular, hyaline conidia, $10–14 \times 4–6\,\mu$.

The complete life-history was first worked out by Klebahn[48].

This fungus affects the leaves and twigs of plane trees, frequently causing considerable defoliation in early summer. The mycelium chiefly occupies the leaf tissues in the vicinity of the main veins, causing them to become brown, but it may spread into the leaf stalk. Acervuli are formed on the under surface of the leaves. Young twigs are also attacked and sometimes killed; in these the conidial stage, formerly known as *Discula*

Platani, Sacc., with unicellular, hyaline spores, is produced. These conidia are one of the means of re-infection in the spring. Perithecia are formed in over-wintered leaves lying on the ground.

Gnomonia leptostyla, (Fr.) Ces. and de Not.

This fungus, the conidial stage of which was called *Marssonnia Juglandis*, (Lib.) Magn., produces brown blotches on walnut leaves. Perithecia are formed on over-wintered leaves.

Gnomonia Rubi, (Rehm) Winter

Perithecia with a long, slender beak ; asci 5–8 spored, usually 6-spored, 4 spores being larger than the other 2 ; the 4 large asco-spores are divided into 2 equal cells with rounded ends, the smaller spores being unicellular or uniseptate.

A die-back of rambler roses ('American Pillar', 'Minnehaha', and 'Lady Gay') caused by this fungus has been investigated by Dowson[40]. The same fungus occurs on the bramble and loganberry. Dowson considers that infection of rambler roses chiefly takes place by ascospores germinating on buds which have been killed by spring frosts. The mycelium penetrates into the wood, passing upwards and downwards. The leaves become yellow and the shoots die back to the ground. Perithecia are formed on the dead twigs during the winter and discharge their ascospores in the spring, the asci moving up to the ostiole, one at a time, before ejecting the spores.

Rehmiellopsis, Bubak and Kabat

Perithecia as in Gnomonia, but they are not beaked and the asci are many-spored.

Rehmiellopsis bohemica, Bub. and Kab.

This fungus is harmful to the foliage and young stems of the common silver fir and other species of Abies in Bohemia and Scotland. According to Wilson and Macdonald[50], young trees are attacked with great severity, the leaves of the current year reddening and shrivelling soon after expansion. Badly affected shoots usually die. The mycelium permeates the whole of the leaf and forms pycnidia with oval, hyaline spores, and then perithecia. Each ascus contains 10–24 uniseptate, hyaline

spores, the upper cell being broader than the lower; the asco-
spores measure 10–21×4–$6\,\mu$.

Glomerella, Spaulding and von Schrenk

Perithecia caespitose, membranous, flask-shaped, dark brown, more
or less immersed in a stroma; asci clavate, 8-spored; ascospores
oblong, slightly curved, unicellular, hyaline.

Conidial stages of the Gloeosporium or Colletotrichum type
commonly occur.

Glomerella cingulata, (Stoneman) Sp. and von S. ($= G.$ *rufo-maculans*, (Berk.) Sp. and von S.).   Bitter Rot of Apples.

Perithecia more or less grouped together; asci evanescent, 55–$70 \times 9\,\mu$; ascospores curved, 12–22×3–$5\,\mu$.

Conidial stage $=$ *Gloeosporium fructigenum*, Berk.; conidia
oblong, unicellular, hyaline, 12–16×4–$6\,\mu$.

Cultural studies by Shear and Wood [51] indicate that many forms
of Gloeosporium and Colletotrichum are probably identical with
this fungus.

This fungus causes a rot of apple fruits and a canker of the
twigs; it also affects other hosts, e.g. pear, peach, grape, orange,
mango, hawthorn and privet. In apple cultivation it is of
greatest importance in the eastern United States, where in
warm, wet summers heavy losses may be caused by it. In
Britain the fungus is commoner on fruit in storage than on
the tree, although it is generally infrequently seen. On the
fruit the fungus produces small brown spots rather late in the
season, which spread until the whole fruit may be involved.
The affected parts sink, and on them appear the conidial pus-
tules. Perithecia occur occasionally on the fruits, and they
have been obtained in culture. Young fruits mummified by
the fungus hang on the trees during the winter and are a
source of re-infection. Spaulding and von Schrenk [52] have
shown that short-lived cankers are formed by the fungus on
the branches, which produce conidia early in the summer.

The disease, where liable to be serious, can be controlled by
the destruction of the mummified fruits and cankers, and by
spraying the trees with Bordeaux mixture, beginning about
a month after the blossoms have fallen.

Glomerella Gossypii, (South.) Edg.   Cotton Anthracnose.

This species is thought to be identical with *G. cingulata* by Shear and Wood[51], but Edgerton[53] considers it distinct.   The conidial stage was formerly known as *Colletotrichum Gossypii.* This is the only spore stage usually seen on the cotton plant. The conidia are formed in acervuli, which are interspersed with sterile setae.

All aerial parts of the cotton plant may be attacked, but the disease is most serious on the seedlings and the bolls.   The fungus is often carried over on the seed, from which the seedlings become affected and then wilt.   Upon the bolls the fungus produces red spots which rapidly enlarge, turn black, and become depressed.   In partly affected bolls the fungus may grow inwards and contaminate the seed.   Lesions also occur on the stems and leaves, sometimes producing a scald effect on the latter.

According to Lehman[54] the fungus on the seed can be destroyed, without serious reduction of germinability, by subjecting the seed to 24 hours' desiccation at 60° C. followed by 12 hours at 95° C.

Valsa, Fries

Perithecia in a more or less definite stroma, black ; asci clavate or cylindrical, often long-stalked, 8-spored ; ascospores unicellular, rarely 2-celled, cylindrical, rounded, slightly curved, hyaline or rarely light brown ; paraphyses absent.

Pycnidial stages of the Cytospora type commonly occur.   Many forms of Cytospora, however, have not yet been adequately related to perithecial stages.

The genus is often sub-divided into sub-genera, e. g. Eutypa, Eutypella, &c.

Valsa leucostoma, (Pers.) Fries

Stroma convex, whitish within, 2–3 mm. across ; perithecia immersed ; asci fusoid-clavate, 35–45×7–8μ ; ascospores biseriate, curved, hyaline, 9–12×2–$2\cdot5 \mu$.

Pycnidia ($=$ *Cytospora leucostoma,* Sacc.), formed in a stroma, many-chambered, conidia extruded in reddish tendrils; conidia curved, hyaline, 5–$6 \times 1 \mu$.

Stone fruit trees often die back in association with the presence of various fungi, one of which is this species.   At first

usually a single limb shows yellowish foliage, which wilts and turns brown, and then the branch dies. Gum often exudes in large masses at the base of the dead branches. The disease often affects the main stem, when the entire tree dies as soon as the bark is girdled. The mycelium permeates the wood as well as the bark. On the dead bark there arise minute swellings from which exude slender reddish tendrils of spores formed in pycnidia embedded in the tissues. At a later stage densely crowded perithecia are formed in the dead bark.

The disease occurs wherever stone fruits are grown, but there is much uncertainty as to the exact mode of infection. In general, trees are attacked by this fungus only when weakened or injured in some way. Infection may arise through wounds, through twigs killed by frost, or, according to Cunningham [55], in consequence of spray injury. Trees planted in soils that are too wet or too dry often succumb to this fungus. Briton-Jones [56] considers that 'die-back' of stone fruit trees is primarily a non-parasitic disorder in which the associated micro-organisms merely behave as weak parasites on tissues gravely weakened by other causes. In the author's opinion, on the contrary, much of the 'die-back' seen in vigorous plum plantations in England in recent years is due to parasitic agency. Aderhold [57] considered that *V. leucostoma* behaved as a wound parasite in cherry trees, and Rolfs [58] established the disease artificially in the peach, apricot, and cherry. Wormald [59] also investigated a similar disease in cherries, associated with *Cytospora leucostoma*, but failed to reproduce the disease by inoculation.

Several other species of Cytospora are associated with the dying-back of stone fruit trees, but these have not yet been sufficiently investigated. Some of these species of Cytospora have been described by Belgrave [60]. Wiltshire [61] found that a Cytospora associated with a 'die-back' of plum trees was always accompanied by a bacterium. The author has also found a bacterium almost constantly present, in association with various fungi, in the 'die-back' disease* of plum trees

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* Dr. H. Wormald, of the East Malling Research Station, who is investigating this 'die-back', showed members of the British Mycological Society,

that has been very prevalent in England in recent years. In this disease the main stem is most commonly attacked, and though death usually ensues in consequence of the bark being girdled, a canker-like lesion is sometimes formed instead.

Hubert [62] states that *C. chrysosperma*, (Pers.) Fr., is pathogenic to poplars and willows in the United States.

Diaporthe, Nitschke

Perithecia embedded in a stroma of variable form, with long, filiform necks, black ; asci cylindrical or clavate, 8-spored ; ascospores elliptical or spindle-shaped, with a median septum, hyaline ; paraphyses absent.

Pycnidial stages, including Phomopsis and Cytospora forms, occur.

Diaporthe perniciosa, E. and E. Marchal

Perithecia formed in a stroma, often grouped together, black, with protruding necks, 1–3 mm. long; asci 50–$60 \times 8 \mu$, 8-spored ; 2-celled, hyaline, 10–$17 \times 2 \cdot 5$–5μ.

Pycnidia formed in a stroma, black, often multilocular, and containing 2 types of spores : ' a ', unicellular, hyaline, fusiform, or elongate oval, 7–9×2–3μ ; 'b ', unicellular, hyaline, filiform, usually hooked at one end, 25–$28 \times 1 \mu$.

This fungus was first described in Belgium by E. and E. Marchal [63] as the cause of a rot of ripe and unripe fruits of apples, pears, plums and cherries, and a canker of these trees. Cayley [64] has since found this fungus to be the cause of a dieback of stone fruit trees, pycnidia being first formed in the dead bark and then perithecia. The pycnospores exude as long, whitish tendrils. According to Briton-Jones [56] this species is sometimes merely a weak parasite capable only of attacking trees enfeebled by some other cause. Deighton [65] found that *D. perniciosa* was non-pathogenic when inoculated into vigorous plum branches. Kidd and Beaumont [36] point out that this fungus often causes a late storage rot of apples.

in July 1927, plum and cherry trees affected by this disease after inoculation with a culture of a bacterium isolated from plum trees attacked by ' dieback '. This bacterium also affects the leaves, producing a ' shot-hole ' effect.

Diaporthe batatis, (E. & Hals.) Harter and Field

This species causes a storage dry rot of sweet potatoes in the United States, and it is also found on the stems and leaves in the field. Perithecia have only been seen in culture, the pycnidial stage (*Phomopsis batatis*) being responsible for the spread of the disease. Like other storage rots of sweet potatoes, damage by this fungus can be largely prevented by storage at a relatively low temperature in a dry atmosphere.

Diaporthe taleola, (Fr.) Sacc. (= *Aglaospora taleola*, Tul.)

This fungus causes a bark canker of oak trees under 40 years of age, although the cankers rarely girdle the branches. The perithecia are grouped together, three of them usually having a common neck ; the ascospores bear 5 thread-like appendages. Sickle-shaped conidia also occur.

Botryosphaeria, Ces. and de Not.

Perithecia embedded in a black stroma which is ultimately erumpent ; asci club-shaped, 8-spored ; ascospores elliptical or egg-shaped, unicellular, hyaline ; paraphyses present.
    Conidial stages occur.

Botryosphaeria Ribis, Grossenbacher and Duggar

A wilt and die-back of red currants, similar to that due to *Nectria cinnabarina* in Europe, is caused by this fungus in the United States, where the disease has been investigated by Grossenbacher and Duggar [66]. Pycnidia and perithecia, formed on the dead twigs, liberate their spores in early summer.

Dibotryon, Theissen and Sydow

Perithecia as in Botryosphaeria, but the ascospores are 2-celled.

Dibotryon morbosum, (Schw.) Theiss. & Syd. (= *Plowrightia morbosa*, (Schw.) Sacc.) Black Knot of Cherries and Plums.

Stromata elongate, cushion-shaped, erumpent, black, up to 25 cm. long ; perithecia scattered ; asci clavate, 8-spored ; ascospores unequally 2-celled, hyaline, $16–20 \times 8–10 \mu$; paraphyses filiform.
    Unicellular, light brown conidia are formed on the surface of young stromata.
    This species has recently been made the type of a new genus by Theissen and Sydow [67].

This fungus causes the ' Black Knot ' disease of wild and cultivated plums and cherries in N. America, but it has not yet been reported in other countries. The fungus infects the fruit spurs and twigs, penetrating to the cambium. Occasionally the whole of the bark may be permeated by the mycelium,
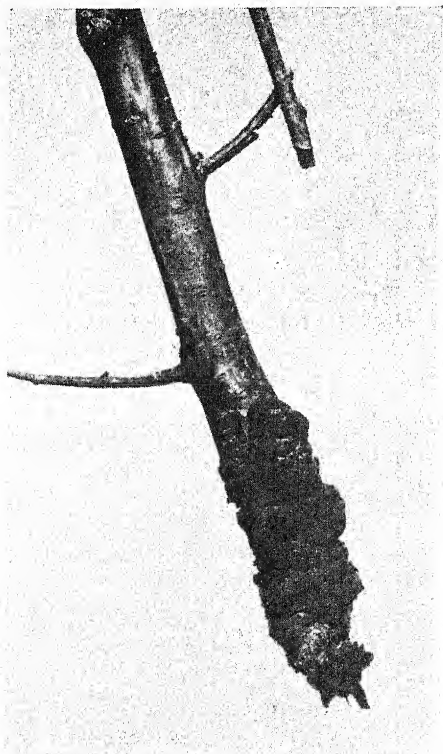
Fig. 37. *Dibotryon morbosum* stoma on *Prunus americana.* Natural size.

when the twig dies. Large elongated knots of fungal tissue are formed in the bark ; these emerge as yellowish bodies, which become black and form perithecia during the summer.

The disease may cause serious losses. Owing to its presence on wild trees it cannot be eradicated, although it can be kept under control by excising the young knots in the early winter.

Endothia, Fries

Stromata finally erumpent ; perithecia brownish-black or black, with long necks ; asci cylindrical, 8-spored ; ascospores spindle-shaped or elliptical, 2-celled, hyaline ; paraphyses absent.

Pycnidia of the Cytospora type usually precede the formation of perithecia.

Endothia parasitica, (Murr.) Ander. and Ander.   Chestnut Blight.

Perithecia aggregated in clusters of 10–20 ; asci 45–$50 \times 9\,\mu$; ascospores 9–10×4–$5\,\mu$.

Pycnospores exuded in yellowish tendrils, slightly curved, hyaline, 2–$3 \times 1\,\mu$.

This disease has almost completely destroyed the chestnut trees (*Castanea dentata*) in the forests of the eastern United States since 1904, when it was first noticed. The fungus is indigenous on other species of chestnut in China and Japan, where it does little harm. It was probably brought into the United States with the introduction of oriental species of chestnut.

The fungus can only invade the twigs and branches through wounds. The mycelium discolours and kills the bark and sap wood, with the result that the parts above the attacked region die. When branches die during the summer the leaves wilt, turn brown, and remain hanging on the tree. In the course of a few years the whole of the tree usually dies. With actively growing trees the growth of the fungus in the bark may be temporarily stayed by the formation of a canker. Pycnidia and perithecia are formed in enormous numbers in the dead bark. The disease has been investigated by Anderson and Rankin [68] and others.

Strenuous efforts have been made to prevent the spread of this disease, especially in Pennsylvania, but without avail. The spores are spread by the splashing of rain, by insects and by wind.

Other species of Castanea are less susceptible to attack, and certain races of the Japanese chestnut (*C. crenata*) are markedly resistant.

Nummularia, Tulasne

Stroma superficial, crust-like, orbicular, black; perithecia peripheral, immersed; asci cylindrical, 8-spored; ascospores sub-elliptical, brownish black; paraphyses filiform.

Conidia are formed just under the surface of the young stroma.

Nummularia discreta, Tul. Blister Canker of Apple.

According to Hasselbring [69] this fungus causes a serious canker of apple trees in some parts of the United States, where it also occurs as a saprophyte on other trees. It has been recorded on the continent of Europe, but not in Britain. In the early stages the cankers exhibit a mottled appearance owing to the inclusion of bits of healthy bark in the diseased area. The older cankers are much cracked and blackened. The circular stromata of the fungus are attached to the wood, but protrude through the bark as blister-like bodies. The perithecia are embedded in the stromata. The ascospores are brown, nearly spherical, and measure $13 \times 10\,\mu$. The fungus is a wound parasite, pruning snags being a frequent source of infection.

Ustulina, Tulasne

Stroma superficial, effused, irregular in shape, at length hard and black; perithecia peripheral, immersed; asci cylindrical, 8-spored; ascospores spindle-shaped, unicellular, black.

Conidia are formed on the surface of the young stroma.

Ustulina zonata, (Lév.) Sacc.

Stroma often zoned, sometimes 10 cm. across, 3 mm. thick, at first white, then dirty grey, finally blackish, dotted over with the perithecial ostioles. Perithecia globose; asci 8-spored; ascospores spindle-shaped, somewhat inequilateral, black, $28-32 \times 7-10\,\mu$; paraphyses present.

In the white stage the stroma abstricts unicellular, hyaline conidia, $6-8 \times 2-3\,\mu$.

Van Overeem [70] considers this species to be identical with *U. maxima*, (Weber) von Wettstein (= *U. vulgaris*, Tul.).

This fungus causes a serious root disease of tea in Ceylon and of rubber in Malaya. It has been investigated by Petch [71], Brooks [72], and Sharples [73].

U. zonata is a common saprophyte in the tropics, especially on the dead stumps of jungle trees and of trees originally planted for shade in tea plantations, but subsequently felled. The mycelium passes along the lateral roots of the stump and may pass thence into the roots of tea bushes or old rubber trees. The mycelium then grows back through the tea or rubber roots to the collar; at the same time the foliage becomes thin, the branches die back, and the bush or tree may die, although one side of an affected rubber tree may continue to yield latex after the other side is dead. Fructifications are produced around the collar or higher up the tree. The wood and the bark are permeated by the mycelium, which forms irregular black lines in the tissues. The mycelium does not spread independently in the soil and is not visible on the surface of the roots.

In rubber plantations the fungus may affect the trunk and branches as well as the roots, chiefly in connexion with attack by boring beetles. These insects often attack the trunks of trees after thinning-out operations, necessitated by too dense planting, or when the trees have been scorched by fire. The felled trees, if left lying about in the plantations, afford an excellent breeding-ground both for the beetles and for *U. zonata*, and the latter often follows an attack of standing trees by the former.

Owing to attack by this and other root-invading fungi it is now customary to uproot and burn all jungle stumps during the early life of a rubber plantation. With tea also the stumps should be removed, including those of shade trees that are cut down. The trees felled during the thinning-out of a rubber plantation should be burnt immediately; care should be taken in burning them not to scorch the bark of the trees that remain.

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CHAPTER XIII

FUNGUS DISEASES (*continued*): USTILAGINALES

USTILAGINALES

THE mycelium in the host ultimately forms masses of spores (brand spores), which are generally dark-coloured and which usually produce basidia (pro-mycelia) on germination. The basidiospores (sporidia), or conidia budded from them, often fuse in pairs. Infection of the host is brought about either by the 'basidium' without formation of sporidia, or by the germ-tube arising after fusion of two sporidia (or conidia), or by a hypha formed after fusion of two hyphae derived from separate sporidia, or by the germ-tube of another spore formed on a mycelium which has arisen after fusion of two hyphae (cf. Dickinson [1]).* Many species comprise several biologic forms (physiological races or varieties).

These fungi, known familiarly as Smut Fungi, often produce no adverse effect on their hosts until spore formation. Some have recently been cultivated on artificial media from brand spore to brand spore by Sartoris [2] and others.

The Ustilaginales of Switzerland have been monographed by Schellenberg [3], and those of Finland are being exhaustively treated by Liro [4].

Ustilago, Persoon

Masses of brand spores black and dusty at maturity; germination usually by the formation of a short, 3-septate basidium, which produces basidiospores (sporidia) laterally.

Ustilago Avenae, (Pers.) Jens. Loose Smut of Oats.

Brand spores formed in the grain and chaff, blackish in mass, becoming powdery; spores spherical to sub-spherical, minutely echinulate on one side, brown, 6–8 μ.

Loose Smut of oats is widely distributed. In a diseased plant the whole of each spikelet, with the exception of the axis, is transformed into brand spores, which may be blown away at maturity. The smutted spikelets, however, may remain intact until threshing, and it is then difficult to distinguish this disease from Covered Smut of oats.

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* Dickinson [1] states that in Covered Smut of oats infection occurs only when mycelia of both sexes are used for inoculation.

The disease is seed-borne; the spores, which often lodge within the glumes, germinate with the grain and thus bring about infection of the seedlings. According to Zade[4a] the spores may also germinate on the glumes before threshing, producing a mycelium bearing 'gemmae', which may subse-
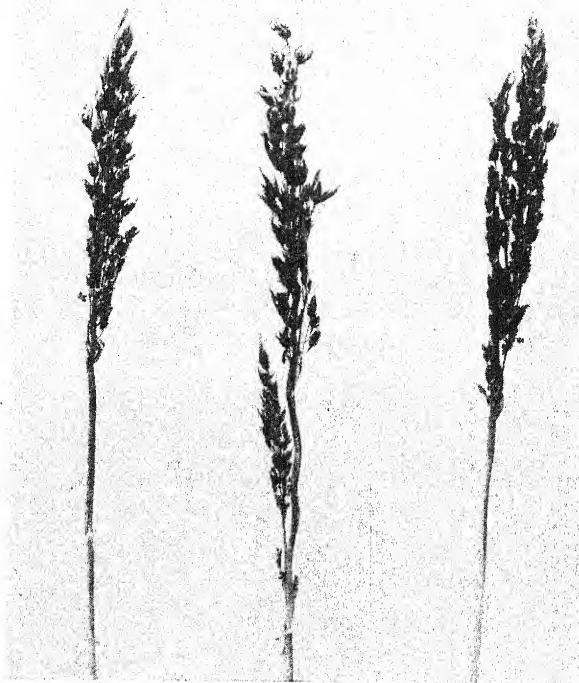
Fig. 38. Oat ears affected by *Ustilago Avenae*. Natural size. (*G. H. Pethybridge*.)

quently infect the seedlings. Once the seedling has been infected the mycelium passes into the stem apex and usually grows up with it. When the inflorescence is formed, the developing grain becomes filled with mycelium which breaks up into masses of brand spores. Groups of brand spores may be formed occasionally on the leaf sheath which surrounds the ear before emergence. Healthy grain may become contaminated by wind-borne spores. During threshing also, smutted ears,

from which the spores have not blown away, may be the means
of dusting the grain with spores. The smut spores do not
retain their vitality in ordinary soil for more than a few
weeks. Comparatively low soil temperatures favour infection,
and white oats are usually less susceptible than black oats.

Infection of the seedling does not always result in a smutted
inflorescence, for the primary axis may sometimes grow faster
than the mycelium, which then remains in the lower part of
the plant. The author has frequently seen fields of smut-free
oats that after harvest have thrown up weak tillers with
small ears 90 per cent. of which have been smutted. A possible
explanation of this is that the crop was more or less uniformly
infected by the fungus in the seedling stage, but the mycelium
failed to keep pace with the development of the primary axes ;
when the late tillers were produced after harvest, the mycelium
advanced into these and the ears became smutted.

Most varieties of *Avena sativa* are susceptible to loose smut,
but *A. strigosa* is immune from or very resistant to it. Reed[5]
and Sampson[6] have shown that biologic forms of *U. Avenae*
exist, the form commonly occurring in Wales being different
from that in Missouri. Further investigation may show that
distinct forms of Loose Smut occur in the same locality.

Loose Smut of oats can be prevented by treating ('pickling')
the grain as follows with formalin just before sowing, the
formalin killing the spores adhering to the grain without
injuring it :

The oats are heaped on the barn floor and are sprinkled with
weak formalin (1 pint of commercial formalin to 40 galls. of
water, or $\frac{1}{2}$ fluid oz. formalin per gall.) at the rate of about $1\frac{1}{2}$ gall.
per sack of grain. The oats are turned over until completely
wetted. The grain should be covered with sacks, also moistened
with weak formalin, for 4 hours. The grain is then spread out and
allowed to dry overnight, when it is ready for sowing.

Alternatively, the oats may be steeped in formalin for 15 mins.,
then heaped on the floor, covered with sacks, and treated subse-
quently as above.

If the farmer can be certain of obtaining seed grain from
fields entirely free from smut and uncontaminated in threshing,
'pickling' is unnecessary.

Ustilago Kolleri, Wille (= *U. laevis,* (Kell. and Swingle) Magnus)
Covered Smut of Oats.

The brand spores are formed in the grain, the chaffy scales being
more or less unaffected and the spore mass remaining compact;
the spores are similar to those of *U. avenae,* but the walls are
smoother.

Covered Smut of oats is believed to be much less common
in Britain than Loose Smut, but it is prevalent in the United
States. There is sometimes great difficulty in distinguishing
Covered Smut from Loose Smut of oats. In Britain varieties
of *Avena sativa* are resistant to Covered Smut, but varieties
of *A. brevis* and *A. strigosa* are susceptible. The life-history
of the fungus is essentially the same as that of *U. Avenae,* and
it can be prevented in the same way.
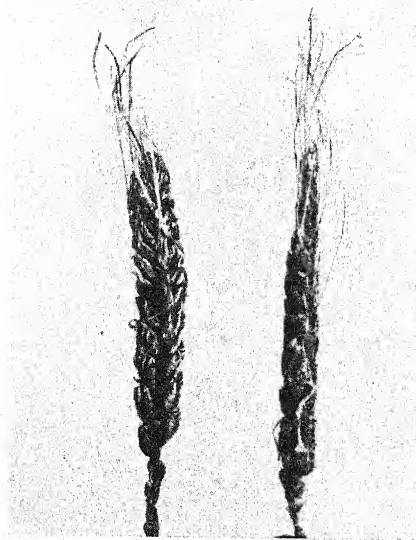
Fig. 39. Barley ears affected by *Ustilago Hordei.* Natural size.

Ustilago Hordei, (Pers.) Kell. and Swing.   Covered Smut
of Barley.

Brand spores formed in the grain, remaining covered by the basal
parts of the glumes, blackish in mass; spores spherical or some-
what angular, smooth, brown, 6–7 μ.

This smut is often present to a serious extent in barley. Smutted ears sometimes imperfectly emerge or fail to emerge from the sheath. In severely infected plants the tips of the upper leaves may also show smut pustules. All the grains of a diseased plant are usually transformed into masses of brand spores, but the chaffy scales, although modified, form a semi-transparent covering over the spores, so that these are not blown about until the smutted ears are broken in threshing. At this time healthy grain may become contaminated. Infection is brought about as in the oat smuts, and the development of the fungus in the host is the same as in the latter. Faris [7] has shown that biologic forms of this fungus occur in the United States. The disease can be prevented by treating the grain before sowing with formalin as in the treatment for oat smut.

The fungus occurs also on wild species of Hordeum.

Ustilago nuda, (Jens.) Rostrup   Loose Smut of Barley.

Brand spores formed in the grain and chaff, blackish in mass, becoming powdery; spores spherical to sub-spherical, minutely echinulate, brown, 5-8 μ.

Loose Smut of barley is much less common in England than Covered Smut. The spores are blown about freely at maturity, leaving the stalks of the ears bare. The ears of smutted plants emerge a few days before those of healthy ones. Occasionally the upper leaves are smutted also. Infection of the grain is usually brought about by smut spores alighting and germinating on the stigmas of healthy flowers. The germ-tubes, which do not form sporidia, grow from the stigmas into the developing grains, but do not prevent the complete formation of the latter, so that diseased grains cannot be distinguished from healthy ones at harvest. The mycelium in the embryo remains dormant until the grain is sown, when it reawakens to activity and infects the growing-point of the seedling shoot, after which the development is the same as in *U. Hordei*. Varieties of barley the flowers of which remain open the longest time, are most liable to attack by this smut.

Where flower infection occurs, treatment of the grain with

formalin is useless, as this substance does not reach the mycelium within the grain. The disease can be prevented, however, by steeping the grain in water at a temperature of 124° F. (51° C.), which is sufficient to kill the mycelium without seriously affecting the germination of the grain. The method of procedure is as follows:

The grain, placed in a sack, is steeped for 4 hours in cold or lukewarm water. It is then plunged in water at 120° F. (49° C.) for 5 min., and finally steeped in water at 124° F. (51° C.) for 10 min.

The temperature must not be allowed to rise as high as 130° F. or serious damage to the grain may ensue. The grain is then laid out to dry and sown as soon as possible.

Investigations by Tisdale and Tapke [8] and others indicate that infection of the seedlings by spores adhering to the grain may also occur, and if this proves to be common it may perhaps be worth while to treat the grain with formalin before sowing (see p. 214), where the hot water treatment is too troublesome to adopt.

Crops of barley affected by Loose Smut should not be retained for seed purposes.

Ustilago Tritici, (Pers.) Jens.   Loose Smut of Wheat.

Brand spores formed in the grain and chaff, blackish in mass and becoming powdery; spores spherical to sub-spherical, minutely echinulate, brown, 5–8 μ.

The life-history of Loose Smut of wheat is essentially the same as that of Loose Smut of barley. The smutted ears become conspicuous as they emerge from the leaf sheaths. The spore masses soon become loose and powdery, and the spores are readily blown about by wind, leaving the bare axes of the ears. Infection of the developing grain occurs at the time of flowering. The promycelia arising from brand spores, which have alighted on the stigmas, usually cause infection directly without forming sporidia. Grain containing mycelium cannot be distinguished superficially from healthy grain. Infection is most common in those varieties of wheat the flowers of which open most widely and for the longest time. The mycelium in the embryo can be destroyed or so weakened

as to be rendered innocuous by steeping the grain in hot water as described for Loose Smut of barley ; the temperature of the water in this case should be between 125° and 129° F. (52°–54° C.). Owing to the troublesome nature of this treatment every effort should be made to obtain seed from healthy fields. It is not yet known whether infection of the seedling may be brought about by spores adhering to the grain.

The species of Ustilago causing the Loose Smut of rye is identical with this fungus, as Humphrey and Tapke [9] have shown by cross-inoculations.

Ustilago Zeae, (Beck.) Ung.　Maize Smut.

Brand spores produced in swellings on any part of the shoot of the host ; spore masses brown, becoming powdery ; spores elliptical to spherical, echinulate, brown, 8–11 μ.

Infection of any part of the actively growing maize shoot may be brought about by sporidia formed after the germination of the brand spores. The germ-tubes grow directly through the epidermis. Adventitious roots above ground may also be attacked. The mycelium is always localized, and associated with its development there is pronounced hypertrophy of the host tissues, resulting in the formation of large galls, which may arise on the leaves, stems, or inflorescences. In smutted plants the male inflorescence may bear female or hermaphrodite flowers in its lower parts. Under suitable conditions the smut spores may germinate directly, or they may remain dormant in the soil for a year or more. The fungus may grow saprophytically in manure for a considerable time. Where the crop is being grown for grain, affected stalks should be cut down and burnt before the galls burst and the spores become distributed. Although the disease is usually sporadic, losses up to 25 per cent. may be caused by it.

There are several biologic forms of this fungus, and Stakman and Christensen [9a] state that sometimes infection is only brought about by combinations of certain pairs of these forms.

Ustilago reiliana, Kuehn

This smut affects chiefly the maize inflorescence, but its presence does not result in the formation of galls. It occurs also on

Sorghum. Except in Australia and some parts of India it is comparatively rare.

Ustilago Sacchari, Rabenh.   Sugar-cane Smut.

Sugar-cane Smut occurs in India, Africa, and the West Indies. Affected plants give rise at their tops to long, curved, black shoots, devoid of leaves, which ultimately expose the spores. If the plants are wholly invaded by the mycelium, secondary shoots arise towards the base, which also may be prolonged into spore-bearing organs.   Infection may take place through hairs on the bud scales, when the disease becomes systemic, or through wounds, when the mycelium remains localized.   The disease may be perpetuated by planting sets containing the mycelium.

According to Butler [10] there are great differences in varietal susceptibility, thin canes being attacked more severely than thick ones.

Diseased canes should be destroyed as soon as the disease is noticed.

Ustilago perennans, Rostrup, has a perennial mycelium in the root-stock of *Arrhenatherum avenaceum*, the spores being formed in the florets.

Ustilago hypodytes, (Schlectendal) Fr., affects *Elymus arenarius*, *Agropyrum repens* and other grasses, sometimes stunting their growth and preventing them from flowering. The spores are formed over almost the entire surface of the aerial stems and occasionally on the leaf sheaths.

Ustilago violacea, (Pers.) Fuckel (= *U. antherarum*, (DC.) Fr.), transforms the anthers of species of Lychnis, Silene, and Dianthus into spore masses.   In female flowers the presence of the fungus stimulates the development of the normally rudimentary stamens, forming spores in them.

Sphacelotheca, de Bary

As in Ustilago, but the masses of brand spores are surrounded by a delicate membrane of sterile fungus cells.

Sphacelotheca Sorghi, (Lk.) Clinton

The Covered Smut of Sorghum occurs commonly wherever the crop is grown, and large losses are caused by it in India.

The majority of grains in an affected ear are usually transformed into elongated cylindrical spore-sacs surrounded at their bases by the unaltered glumes. The stamens are not always involved in the spore-sac. In some varieties of Sorghum the infected grains appear normal, but on being ruptured are found to consist of masses of blackish spores.

The life-history of the fungus is similar to that of *Ustilago Avenae*, and infection is usually brought about during the germination of the grain. According to Brefeld and Falck [11] infection through the flower may perhaps occur rarely. They also showed that when the seedling grows very rapidly the mycelium cannot keep pace with the growing apex, so that the terminal inflorescence remains healthy; if the upper part of such a plant is cut away, lateral inflorescences are produced, which become smutted.

The disease can be reduced greatly by treating the grain before sowing with formalin (see p. 214) or with $2\frac{1}{2}$ per cent. copper sulphate solution in the same way.

Sphacelotheca cruenta, (Kuehn) Potter, causes a Loose Smut of Sorghum.

Tilletia, Tulasne

Masses of brand spores blackish and dusty at maturity; brand spores germinating by means of a short basidium (promycelium), which forms elongated basidiospores (sporidia) terminally; the basidiospores, with or without fusion in pairs, give rise to a germ-tube or a mycelium that forms secondary spores.

Tilletia caries, (DC.) Tul. (= *T. Tritici*, (Bjerk.) Wint.) Bunt or Stinking Smut of Wheat.

Brand spores formed in the grain, more or less concealed by the glumes and pales, blackish and 'oily' in mass, with a fish-like odour; spores spherical, with winged reticulations, brown, $16–22\,\mu$.

Bunt is one of the most serious diseases of wheat; it occurs commonly in all wheat-producing countries. In some seasons thousands of bushels of grain are lost in England in consequence of it. An infected plant frequently shows no obvious sign of disease until the ears emerge, but the stem of a bunted plant is usually shorter than that of a healthy one. Bunted ears

are somewhat narrower than healthy ears, but, as ripening
proceeds, the glumes are pushed farther apart laterally than
usual in many varieties, so that
bunted ears can often be detected
fairly easily. The grain is trans-
formed into a blackish mass of spores,
but this generally remains covered
until threshing. All the ears of an
infected plant and all the grains in
the ear are usually bunted. The
spore masses are more or less olea-
ginous when fresh, and have a fish-
like odour, hence one of the names
given to the disease. During thresh-

Fig. 40. Germinating brand
spore of *Tilletia caries*, × 400.
(*R. W. Marsh.*)

ing many of the bunted grains are broken, and so the healthy
grain becomes dusted with spores, sometimes to such an extent
as to appear blackish. The threshing-machine may also become
contaminated and may be the means of contaminating healthy
wheat subsequently threshed in it.

Infection occurs during the seedling stage, the spores on the
grain germinating at about the same time as the grain does.
The coleoptile is penetrated by germ-tubes formed after fusion
of the sporidia in pairs; the mycelium then passes into the
young stem and keeps pace with the growth of the apex until
the inflorescence is formed. There is some reason for believing
that when wheat, infected with the bunt fungus, grows par-
ticularly well in the spring, the stem apex may sometimes
outpace the growth of the bunt mycelium, with the result that
the ears are not attacked.

Under British conditions the brand spores do not retain
their vitality in the soil for any length of time, but in some of
the Pacific North-west States of the United States infection
from spores in the soil is frequent, owing to the practice of
sowing winter wheat on fallow land directly after the soil has
become contaminated by bunt spores liberated in threshing.

In England, winter wheat is usually more affected than
spring wheat, but most of the varieties commonly grown
in this country are susceptible. Comparatively low soil

temperatures appear to favour infection.   In the United States
the varieties 'Turkey', 'Kanred', 'Sherman', 'White Odessa',
'Ridit', 'Martin', and 'Hussar' are markedly resistant to Bunt,
except to certain particular biologic forms (see under *Tilletia*
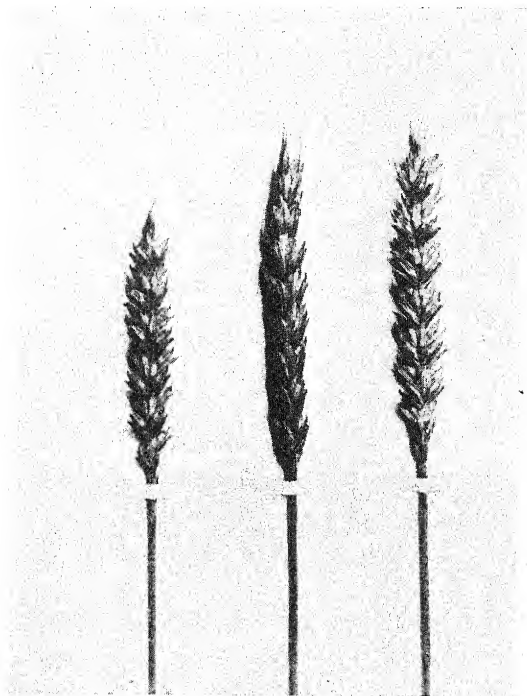
FIG. 41. Bunted ears of ' Little Joss ' wheat, healthy ear in middle.
$\frac{1}{2}$ natural size. (*A. Smith.*)

levis).   These are now being tested for resistance to Bunt in
England, but so far only the varieties ' Martin ' and ' Hussar '
have proved very resistant here.*   Attempts are being made
to combine the quality of resistance to Bunt shown by these
varieties with the other desirable qualities of English wheats.
In the United States Gaines [12] has studied the genetics of bunt

* Information kindly supplied by Mr. W. A. R. Dillon Weston, School of
Agriculture, Cambridge. Miss Sampson has also found that ' Martin ' and
'Hussar' are very resistant to Bunt (*Welsh Journal of Agriculture*, vol. iii, 1927)

resistance, and has shown that in some of the descendants of crosses between two resistant types the degree of resistance is considerably enhanced. Briggs [13] states that resistance to Bunt is dominant in the F_1 hybrids between certain susceptible and resistant varieties.

Bunt is of such common occurrence at the present time that no reliance can usually be placed upon seed wheat being uncontaminated by spores. It can be effectively controlled, however, by treating the grain with formalin as described on p. 214.* Alternatively, a $2\frac{1}{2}$ per cent. solution of copper sulphate can be used for sprinkling or steeping the grain, but under English conditions formalin is usually preferable. In certain countries, e. g. Australia and the United States, where some of the grain is often injured in threshing, the use of formalin may be detrimental to germination. In consequence of this it is becoming customary in those countries, and in others, to dust the grain with powdery copper carbonate or copper acetate before sowing, with which very good results have been obtained. About two ounces of copper carbonate are incorporated with a bushel of grain. In this treatment it is important that the fine powder should be dusted over the whole surface of the grain; this is best done by using a special dusting machine. Other substances, e.g. uspulun, germisan, &c., containing organic mercury compounds, have also been successfully employed in the seed treatment of wheat against Bunt, particularly in Germany.

Lang [14] has pointed out that attack by Bunt increases the susceptibility of wheat to *Puccinia glumarum*.

Tilletia laevis, Kühn

This species is very closely related to *Tilletia caries* and differs from it only in the absence of reticulations on the spores. The life-history is essentially the same as in *Tilletia caries*. *Tilletia laevis* is commoner than *Tilletia caries* in North America, but it rarely occurs in Britain. Rodenhiser and Stakman [15] and Reed [15a] have called attention to the existence

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* One part of formalin in 480 of water is sufficiently strong for the control of Bunt.

of distinct biologic forms in both *T. laevis* and *T. caries*. Reed states that the most useful varieties of wheat for differentiating these biologic forms are Martin, Hussar, White Odessa, Kanred, and Turkey.

Tilletia horrida, Tak.

Bunt of rice is widely distributed in rice-growing countries, but it is rarely serious. Only a few grains in an ear are transformed into spore masses, which remain covered by the glumes. Infection occurs in the seedling stage; the disease can be prevented by pickling the seed in an antiseptic before sowing, although it is generally not worth while to take this precaution.

Urocystis, Rabenhorst

Masses of brand spores blackish and dusty at maturity; spores dark, grouped together in small numbers, each spore ball being surrounded by a number of sterile, lighter coloured cells; brand spores germinating as in Tilletia.

Urocystis occulta, (Wallr.) Rabenh.   Stripe Smut of Rye.

Brand spores formed in the leaves, stem and inflorescences, forming linear striae often of great length; spore balls sub-spherical, 16–32 μ.

The life-history of the Stripe Smut of rye is similar to that of Bunt of wheat, but the masses of brand spores are usually found in long lines on the stem and leaves. Diseased plants are often stunted in growth, and ears may not be formed or may be devoid of grain. The disease, which is rare in Britain, can be prevented by seed treatment as in Bunt of wheat.

Urocystis Tritici, Koernicke   Flag Smut of Wheat.

Flag Smut of wheat occurs in India, S. Africa, the United States, and southern Europe, and Noble[16] states that it is one of the most serious diseases of wheat in Australia. Seedling plants are infected through penetration of the coleoptile by germ-tubes of the fungus, the spores of which may adhere to the grain or persist in some soils in a living condition for at least a year. Affected plants show long greyish stripes on the leaves, which become black when the spores are mature and the epidermis is ruptured. Occasionally the

stalks of the ears and the glumes are also discoloured by black
lines of spores. The parasite causes considerable stunting of the
plant and twisting of the leaves, and often prevents the formation
of ears. Even when ears are formed, they are usually empty or
contain only shrivelled grain. There are considerable differences
in varietal susceptibility to this disease.

Urocystis Cepulae, Frost   Onion Smut.

Brand spores formed in the leaves, especially in the leaf bases,
and in the scale leaves of the bulbs; spore balls sub-spherical,
17–25 μ.

Onion Smut occasionally attacks leeks and shallots as well
as onions. It is an important disease in parts of the United
States, but, so far, it has been recorded at only a few centres
in Britain. Infection of the cotyledon occurs from spores in
the soil. The first sign of attack is a darkening of the base of
the first leaves, in which long black masses of brand spores
are found; other leaves become similarly affected. Some of
the spore balls pass into the soil, which may remain contami-
nated for several years.

The relation of soil temperature to infection has been studied
by Walker and Jones [17] in the United States, who found that
where the soil temperature was 29° C. or higher infection
could not take place. This accounts for the 'immunity' of
onions from this disease in the southern part of the United
States, where soil temperatures are high. Walker and Well-
man [18] have further shown that a temperature of 28° C. almost
completely inhibits spore germination and soon kills any germ-
tubes that may be formed.

Transplanted onions can be grown in contaminated soil, as
the plants cannot be infected after the seedling stage. Care
should be taken not to remove to fresh ground leeks, onions,
or other plants grown in contaminated soil, as the spores may
be distributed thereby in particles of soil adhering to the
roots. The disease can be prevented by applying a weak
solution of commercial formalin (1 in 120 parts of water) to
the drills at the time of sowing. This kills the spores in the
drill but does not injure the seed.

The disease is scheduled in England by the Ministry of

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Agriculture under the Destructive Insects and Pests Acts, and is notifiable to the Ministry.

Urocystis Violae, (Sow.) Fisch. v. Waldh.

Violet Smut often affects cultivated violets, producing large swellings on the stems and leaves, which become filled with black spore-balls.

Urocystis Gladioli, (Requien) W. G. Sm.

This fungus, which has been recorded occasionally in Britain, forms black spore masses within the corms of Gladiolus.

Urocystis Colchici, Rab., produces black streaks in the leaves of *Colchicum autumnale, Muscari racemosum* and other related plants, and *Urocystis Anemones*, (Pers.) Wint., causes a smut disease of anemones.

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FUNGUS DISEASES (*continued*): UREDINALES

UREDINALES

The mycelium ramifies in the tissues of the host, and, in most species, ultimately forms teleutospores, which give rise on germination to basidia (pro-mycelia) or which themselves become basidia. The basidiospores (sporidia) infect the host, usually by cuticular penetration, and the mycelium therefrom often first gives rise to aecidiospores (formed in aecidia or caeomata); the aecidiospores in turn produce another mycelium which gives rise often to uredospores before teleutospores are formed; several generations of uredospores may be produced before teleutospores are finally formed. The germ-tubes of aecidiospores and uredospores infect the host by way of the stomata. Spermogonia (pycnidia), abstricting spermatia (pycnospores), often accompany the aecidia, or rarely the teleutospores in species which produce no aecidiospores. The function of these spermatia or pycnospores is obscure, but recent observations by Craigie * indicate that they may play an important part in the life-cycle.

The Rust Fungi are obligate parasites, the mycelium of which is chiefly intercellular and provided with haustoria. The mycelium may cause considerable hypertrophy of the host, which may be either local or which may involve the entire shoot system. Many Rusts are heteroecious, and, in these, the aecidiospores and teleutospores are always formed on different hosts, which are unrelated to each other. Some species are devoid of aecidiospores and uredospores, and in others uredospores are not formed in the life-cycle. Many species of Rust Fungi comprise several biologic forms or physiological races or varieties. It is rather exceptional for these fungi to cause serious damage to their hosts. The spores are commonly yellow, orange, or brown in colour.

The Rust Fungi of Britain have been described by Grove[1], and those of Switzerland by Fischer[2]. Eriksson and Henning[3], Klebahn[4], and Sydow[5] have published detailed accounts of some of these fungi.

Uromyces, Link

Teleutospores stalked, unicellular, with an apical germ pore. Aecidiospores, formed in cup-like aecidia, and uredospores occur in many species.

* Cp. *Nature*, vol. 120, pp. 116 and 765, 1927.

Uromyces Fabae, (Pers.) de Bary   Bean Rust.

Aecidiospores minutely verrucose, yellow, 14–22 μ. Uredospores distantly echinulate, with 3 germ pores, pale brown, 20–30 × 18–26 μ. Teleutospores smooth, brown, 25–38 × 18–27 μ ; pedicels brownish, long.

This fungus occurs on beans (*Vicia Faba*), peas and certain vetches, although the fungus on the latter is probably a distinct biologic form.

Aecidia are of rare occurrence, but uredospores and teleutospores occur commonly later in the season. The teleutospores do not germinate until the spring. In England the uredospores may perhaps retain their vitality over the winter in sheltered situations, and be the means of infection the following season. In some summers the fungus is abundant on beans, but it is rarely harmful.

Uromyces Pisi, Wint.

This is a heteroecious species, the aecidia occurring on *Euphorbia cyparissias*, and the uredo- and teleuto-stages on the pea and *Lathyrus pratensis*. The sporidia infect the Euphorbia, in the root-stock of which a perennial mycelium is established. Shoots arising from such root-stocks are greatly changed in appearance, the leaves being much shorter, broader, and thicker than healthy leaves. The under surface of the leaves is covered with yellow aecidia, the spores from which infect the alternate host plants, producing brown pustules of uredo- and teleutospores. This fungus is very rare in Britain.

Uromyces appendiculatus, (Pers.) Lév.

This rust attacks *Phaseolus vulgaris*, other species of Phaseolus, the cowpea and certain closely related plants. It is of common occurrence in the United States, in the south-eastern part of which it is serious, and in India, but it is comparatively rare in Europe and very rare in Britain. All spore stages are found on these hosts, the aecidia, which are rarely seen, appearing first. Later in the season brown pustules, first of uredospores and then of teleutospores, are formed on the under surface of the leaves. In some countries the uredospores remain viable over the winter. With beans there are considerable differences in varietal susceptibility, some varieties being markedly

resistant. The fungus on the cowpea is probably a distinct biologic form.

Uromyces Trifolii-repentis, (Cast.) Liro

Aecidia, uredo- and teleutospore pustules occur on *Trifolium repens*. Another species, *Uromyces flectens*, Lagerh., having teleutospores only, is found on the same host.

Uromyces Trifolii, (Hedg. f.) Lév.

The brown uredo- and teleutospore pustules occur commonly on clovers, including *Trifolium pratense*, but not *Trifolium repens*. There are no aecidia in the life-history of this species.

Uromyces Onobrychidis, (Desm.) Lév., affects sainfoin. Uredo- and teleutospores only are produced.

Uromyces caryophyllinus, (Schr.) Schroet. Carnation Rust.

Aecidiospores on *Euphorbia gerardiana*.
 Uredospores sparsely echinulate, brownish, 20–35 × 18–25 μ.
 Teleutospores with a flat, hyaline papilla, minutely punctate, chestnut-brown, 20–21 × 18–24 μ; pedicels short, hyaline, deciduous.

Carnation rust occurs commonly wherever carnations are grown on a large scale under glass. It is rare on other species of Dianthus. The aecidial host does not occur in Britain, where the fungus is spread solely by uredospores. The rust appears as brown, pulverulent pustules on the leaves and occasionally on the stems, which consist of masses of uredo- spores sometimes intermixed with teleutospores. It is only when the rust is very abundant that carnation plants appear unsightly or are crippled in growth. By careful attention to watering, so that the spores are not splashed on to healthy leaves, by adequate ventilation so as to ensure a relatively dry atmosphere, and by propagating only from healthy cuttings, the disease can be kept under control. Varieties differ con- siderably in susceptibility, and many have been discarded owing to liability to attack.

Uromyces Betae, (Pers.) Tul. Mangold and Beet Rust.

Aecidispores verrucose, yellow, 16–24 × 16–20 μ.
 Uredospores minutely echinulate, with 2 equatorial germ pores, yellowish-brown, 21–32 × 16–26 μ.
 Teleutospores smooth, with an apical papilla, brown, 22–34 × 18–25 μ; pedicels short, hyaline.

The aecidia first appear on the under surface of the leaves of mangolds and sugar beet, and are followed by uredospore pustules and finally by teleutospore pustules. The teleutospores remain dormant on the dead foliage during the winter, germinating in spring to produce sporidia, which infect the young foliage.

The fungus is most common where mangolds and beet are grown for seed, but it is usually merely sporadic. Only where mangolds are excessively manured with nitrogenous compounds is the rust so abundant as to cause an appreciable reduction in the crop. Lack of potash in the soil appears to predispose to attack.

Uromyces Lilii, (Link) Fckl.

This rust is occasionally found on the leaves of *Lilium candidum* and other lilies, aecidiospores and teleutospores being produced. The latter have characteristic striae on their walls.

Hemileia, Berk. and Br.

Aecidia unknown.

Uredospores borne singly on hyphae which protrude in groups through the stomata, often smooth on one side and echinulate on the other.

Teleutospores as in Uromyces.

Hemileia vastatrix, Berk. and Br.   Coffee Leaf Rust.

This rust is almost universally present on coffee in the Old World, but is unknown in America. It was unrecognized and perhaps did not exist in the coffee plantations of Ceylon until 1869, but after that date it spread epidemically and affected the crop to such an extent that it was one of the causes which led to the abandonment of coffee cultivation in Ceylon and Malaya. Arabian coffee was first abandoned and then Liberian coffee, but more recently Robusta coffee, which is less severely attacked, has been grown successfully in the Dutch East Indies.

The rust appears in the form of yellowish spots on the under side of the leaves, and in severe attacks defoliation leads to a great reduction in crop. Uredospores, which are smooth on one side and echinulate on the other, are produced on these

spots and propagate the disease. The teleutospores were first described by Marshall Ward[6], but they are frequently absent. In the Highlands of East Africa Arabian coffee can still be grown successfully. Dowson[7] has pointed out that the disease is comparatively innocuous there at altitudes of 5,000–7,000 ft., where the temperature and the humidity are not high. At a somewhat lower altitude (4,000 ft.) this rust can be kept under control by spraying with Bordeaux mixture or with a modified form of this in which calcium carbide is used instead of lime, but at still lower altitudes coffee cannot be grown successfully on account of the higher temperature and greater humidity, which are more favourable to the fungus than to the host.

Puccinia, Persoon

Teleutospores stalked, two-celled, each cell having one germ pore. Aecidiospores, formed in cup-like aecidia, and uredospores occur in many species.

Puccinia Chrysanthemi, Roze Chrysanthemum Rust.

This fungus produces uredo- and teleutospores in brown pustules on the under surface of the leaves of Chrysanthemums, uredospores being by far the most abundant. Chrysanthemums grown under glass are commonly affected, but the fungus is less prevalent in England than formerly, probably because resistant varieties are now chiefly cultivated. The plants suffer only when they are very badly attacked. Care in watering to prevent distribution of the uredospores by splashing checks the spread of the disease.

Puccinia Menthae, Pers. Mint Rust.

Aecidia hypophyllous or on the stems; aecidiospores verrucose, yellow, 24–40×17–$28\,\mu$.
 Uredospores echinulate, light brown, 17–28×14–$19\,\mu$.
 Teleutospores with a pale apical papilla, indistinctly verrucose, dark brown, 26-35×19-$23\,\mu$; pedicels longer than the spores, hyaline.

Mint rust commonly occurs wherever mint is cultivated; it occurs also on peppermint and on wild species of Mentha. It was formerly thought that the mycelium producing aecidia

was perennial in the root-stock, but A. Smith * has shown that this view is erroneous. Infection of the rhizomes and stems arises from germinating teleutospores throughout the late autumn and winter; the mycelium so formed becomes widespread in the tissues and proliferates with the developing buds, the invaded shoots being considerably contorted and bearing small yellowish leaves. Infection of this kind often occurs from teleutospores lying on the ground. Yellow aecidia are produced on these shoots in the spring, and the aecidiospores infect the leaves secondarily, on which first uredospores that further disseminate the fungus, and then teleutospores arise.

The disease can be evaded by establishing, from year to year, fresh mint beds from healthy rhizomes. In an old mint bed it is advisable to burn off the mint tops in the autumn with the aid of straw before the teleutospores fall to the ground with the leaves.

Puccinia Violae, (Schum.) DC., occasionally affects cultivated violets, but is much commoner on wild forms. The life cycle is completed on the violet plant, aecidiospores, uredospores, and teleutospores being produced.

Puccinia aegra, Grove, is closely related to the preceding species, but it affects Violas. The mycelium producing aecidia permeates the tissues widely, and shoots affected by it are deformed. The aecidia are formed practically over the whole surface of the stems and the under surface of the leaves. Uredo- and teleutospore stages occur on the same host.

Puccinia malvacearum, Mont.   Hollyhock Rust.

Teleutospore pustules brown, hypopyllous or on the stems or fruits; teleutospores attenuated at both ends, thickened at the apex, yellowish-brown, $35–75 \times 12–26 \mu$; pedicels persistent, of variable length, hyaline.

This fungus was first recorded in 1852 on cultivated hollyhocks in Chili, and was discovered in Europe in 1869, when the cultivation of hollyhocks began to be extensive. After this date it spread rapidly and attacked other European species belonging to the Malvaceae. Teleutospores only are produced;

* In investigations carried out at Cambridge, but not yet published.

if formed early in the season they germinate directly and the sporidia spread the disease rapidly, but if the teleutospores are formed later in the year many of them do not germinate until the following spring. When the teleutospores are germinating the pustules are covered with a delicate bloom. Eriksson [8] has pointed out that if the teleutospores are immersed in water, oidia are sometimes formed on germination.

The disease often occurs epidemically on hollyhocks, and, when severe, causes almost complete defoliation. The under surface of affected leaves is sometimes almost covered with the pustules of the fungus. Good methods of cultivation often enable hollyhocks to be grown comparatively free from the disease.

Puccinia Pruni-spinosae, Pers.   Plum Rust.

Aecidia hypophyllous, pale ; aecidiospores finely verrucose, pale yellow, 16–24 μ.

Uredospore pustules hypophyllous ; uredospores echinulate, pale brown, 20–35 × 10–18 μ, mixed with capitate paraphyses.

Teleutospores pustules hypophyllous ; teleutospores consisting of two globose cells which readily fall apart, densely verrucose, brown, 30–45 × 18–25 μ ; pedicels very short, deciduous, hyaline.

The aecidia, accompanied by brownish spermogonia, are found on *Anemone coronaria,* and the other spore stages occur on the leaves of plum and wild species of Prunus. The underground parts of affected anemones are permeated by a perennial mycelium, so that each spring the leaves of diseased root-stocks produce aecidia, the foliage being malformed. Affected anemones rarely flower, or do so imperfectly. The aecidiospores infect plum leaves, producing small yellow spots, on the under surface of which first uredo- and then teleutospore pustules develop. Uredospores disseminate the fungus rapidly in the summer, and occasionally the fungus may be so abundant as to cause premature defoliation. The teleutospores on germination bring about infection of the anemone ; the mycelium grows down into the root-stock and persists there. In England there is probably an obligate relationship between the phase of the fungus on the plum and that on the anemone.

Although the fungus is of little economic importance, it is

advisable to eradicate diseased anemones in the vicinity of plum plantations.

Puccinia lychnidearum, Link, f. *Dianthi*, Grove

This rust occurs on Sweet Williams and occasionally on other species of Dianthus and on Lychnis. The brown teleutospore pustules develop chiefly on the under surface of the leaves, and are often arranged in concentric zones. The teleutospores germinate directly, the sporidia being the only means of infection. Varieties of Sweet William with dark red flowers appear to be resistant to this rust.

Puccinia Antirrhini, Diet. & Holw., causes serious injury to Antirrhinums in the United States. Brown uredospore pustules and darker brown teleutospore pustules occur on the leaves and stems.

Puccinia Iridis, (DC.) Wallr.

This fungus produces uredospores and teleutospores on the leaves of many species of Iris. Both kinds of spores are brown. The fungus does not usually appear until late in the summer. It is not known whether this species is heteroecious or not.

Puccinia Asparagi, DC.   Asparagus Rust.

Aecidiospores globose, orange, 15–28 μ.
    Uredospores densely echinulate, pale brown, 20–30 × 17–25 μ.
    Teleutospore pustules linear and often confluent, blackish; teleutospores brown, 35–52 × 17–26 μ, pedicels as long as or longer than the spores, hyaline or brownish.

Asparagus rust rarely occurs in England, but soon after its introduction into the United States it became a serious pest in certain parts. All spore forms are produced on the stems or 'leaves' of the asparagus plant, aecidiospores being formed first in the season. The teleutospores germinate in the spring after over-wintering, and the sporidia are the only means of re-infection. Dead asparagus stems should be burnt in the autumn to prevent infection in the following spring.

In the United States the disease is most serious where dews are heavy and frequent, as in parts of California. Some varieties of asparagus are comparatively resistant.

Puccinia Porri, (Sow.) Wint.   Leek Rust.

Uredospore pustules yellowish or reddish-yellow; uredospores delicately echinulate, yellow, 20–30 μ.

Teleutospore pustules blackish-brown; teleutospores smooth, brown, 28–52 × 20–26 μ, pedicels short, deciduous, hyaline.

This rust is fairly common on leeks, especially in the north of England and in Wales, and it occasionally appears on other species of Allium. The teleutospores germinate after the winter, the sporidia infecting the same host. The fungus is most often seen on plants kept for seed purposes.

Puccinia pringsheimiana, Klebh.

This is a heteroecious species in which the aecidia are found on the leaves and fruits of black currants and gooseberries, and the uredospores and teleutospores on various sedges. It occurs on these bushes only where they are growing in proximity to sedges, as often happens in Norfolk. It has been seen most abundantly where cut sedge has been used as a mulch in the plantations. The rust malforms the fruit, so that sedges should not be used for mulching these bushes.

Puccinia graminis, Pers.   Black Rust (Stem Rust) of Cereals and Grasses.

Aecidia hypophyllous and on the fruit, on roundish, thickened spots 2–5 mm. in diameter, cylindrical, white, with a cut margin; aecidiospores smooth, orange, 14–26 μ.

Uredospore pustules chiefly on the sheaths and culms, linear, often confluent, brown; uredospores elliptical or ovate-oblong, echinulate, brown, 22–42 × 16–22 μ, usually with 4 equatorial germ-pores.

Teleutospore pustules often formed on the site of uredospore pustules, chiefly on the culms, forming long black lines; teleuto-spores oblong-clavate, much thickened at the apex, smooth, very dark brown, 35–60 × 12–22 μ; pedicels persistent, brownish, thick, up to 60 μ long.

The Black Rust of cereals is a heteroecious parasite, alternating between cereals and grasses on the one hand and the common barberry on the other. The teleutospores embedded in the straw germinate in the spring, and the sporidia infect young leaves and fruits of *Berberis vulgaris*, on which the

aecidia are produced in the early summer; a few other species of Berberis are occasionally affected by the aecidial stage. The aecidiospores infect cereals and grasses in the vicinity, the culms being most commonly affected. On these, brown pustules of uredospores, often arranged in long lines, are first produced, but teleutospores quickly begin to form in
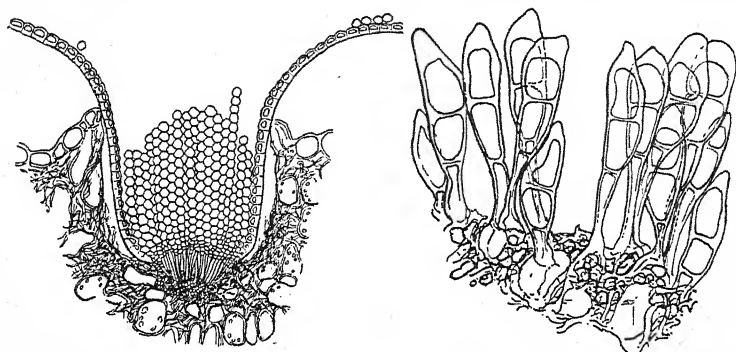
FIG. 42. *Puccinia graminis* : (left) aecidium in section, × 200 (*R. W. Marsh*); (right) teleutospore pustule in section, × 350. (*W. T. Dowson.*)

the same pustules, which, in consequence, turn black. The conspicuous black lines of teleutospores on the culms give the popular name to the disease. The uredospores serve to disseminate the fungus on cereals and grasses. In warm countries the uredospores carry over the fungus from one season to another.

The connexion between the Black Rust or 'mildew', as it was called in the eighteenth century, and barberry bushes, was realized by farmers in England and N. America long before the heteroecious nature of the parasite was fully proved experimentally by de Bary in 1864-5. In 1760 the Legislature of Massachusetts passed a law compelling the eradication of barberry bushes in the neighbourhood of wheat fields. Since then these bushes have been extirpated in the vicinity of wheat in many countries, notably in England and Denmark, and during the last few years the State of Minnesota and neighbouring parts of the United States have voted large sums of money for the destruction of the barberry, which has become naturalized on waste lands there.

Wheat, barley, oats, and rye, are all liable to attack, as well as certain grasses, including couch (*Agropyrum repens*), but it is on wheat that the fungus is most disastrous in its effects. In certain parts of N. America (Minnesota, Dakota, Manitoba, Saskatchewan), Africa, India, and Australia the fungus sometimes causes a 50 per cent. reduction in the crop, and in some places prevents entirely the profitable cultivation of wheat. It is in consequence of the fungus attacking principally the stems that it is often so serious in its effects, for, if the straw is affected before it is hardened, it collapses, with the consequence that the grain does not fill. Where, however, as at present in England, the fungus attacks the crop when nearly mature, if at all, little harm is done.

In N. America the hard red spring wheats, of which 'Marquis' is one of the best known, are most susceptible to attack. Hard red and soft red winter wheats are more resistant. In general, 'vulgare' wheats are more susceptible than 'durum' and 'club' types. Durum wheats are partly replacing the hard red spring wheats in certain areas because of the susceptibility of the latter. In Minnesota, Dakota, and Manitoba the evidence available points to aecidiospores being the primary source of epidemics, for the winters are too cold to allow the uredospores to retain their vitality until the spring. In more southerly parts of the United States the uredospores remain viable over the winter and are a source of infection the following season; in these parts the winter temperatures are sufficiently high to kill the teleutospores so that the barberry does not become affected. In Australia, where aecidia on the barberry are unknown, and in Africa and parts of India uredospores also appear to be the sole means of survival from season to season.

In Britain and northern Europe generally the fungus is only prevalent to a serious extent on cereals where barberry bushes are still abundant. Mehta[9] has shown that in England the intervention of the barberry is necessary for the continued existence of *P. graminis*, for the winter temperatures kill the uredospores.

Puccinia graminis includes several biologic forms or races,

which differ in their powers of infection. Distinct biologic forms may occur in different countries. As regards cereals Eriksson[10] has distinguished the following biologic forms in Sweden:

P. graminis Secalis on rye, barley, *Agropyrum repens*, and some other grasses, but not on wheat or oats.

P. graminis Avenae on oats, *Dactylis glomerata*, and some other grasses, but not on wheat.

P. graminis Tritici only on wheat usually, but Eriksson looks upon this form as being not sharply fixed, as it may feebly infect barley and rye.

The specialization of these forms holds good for aecidiospores as well as for uredospores. Stakman and Levine[11] have shown that there are slight morphological differences between some of the biologic forms of *P. graminis*.

In England, Mehta[9] found that Black Rust of wheat infected wheat and barley, but not rye and oats, and that the form *Secalis* infected rye, barley, *Agropyrum repens*, and one variety of wheat, 'Red Sudan'. The specialization of these forms is of great importance. As an indication of the fixity of this specialization it may be stated that the writer has had under observation for many years a place in which the form *Secalis* is constantly found on *Agropyrum repens*, but he has never seen it affecting neighbouring crops of wheat and oats.

In the United States, Stakman and others[12] point out that *P. graminis Secalis*, after being cultivated for three years on barley, is no more capable of infecting wheat than it was before. As barley is a host for both the forms *Secalis* and *Tritici*, this result confirms the fixity of specialization, and the unlikelihood of the existence of 'bridging hosts' which might enable these forms to pass from one cycle of hosts to another.

In the United States, Stakman and Levine[13] have distinguished more than thirty different biologic forms of *P. graminis* on wheat alone. A variety of wheat may be immune from one or several biologic forms of the rust and susceptible to several others. Several of these biologic forms differ in their geographical distribution. One of these forms

differs considerably from the others in the character of its uredo pustules and spores, and has been distinguished under the name *P. graminis Tritici-compacti*. Some other forms occurring on wheat also show slight morphological differences. Bailey [14] has distinguished five different forms of *P. graminis Avenae*.

As regards the nature of the susceptibility or resistance to *P. graminis* exhibited by different varieties of wheat there is little certain knowledge. According to Stakman [15], when some immune varieties are inoculated with the fungus, penetration at first proceeds in a normal manner, but the fungus quickly kills the surrounding host cells, so that it cannot proceed farther; in a susceptible variety, on the other hand, the fungus on entry establishes haustoria in the host cells, which continue to live and provide food for the further growth of the fungus. Immunity in such cases is due to a kind of 'hypersensitiveness' on the part of the host. A high degree of resistance is associated with similar, but less clearly defined, reactions. Allen [16] has worked out in detail the reaction of 'Mindum' wheat to one form of *P. graminis Tritici* from which it is immune. In this, the first haustorium established by the infection hypha immediately becomes surrounded by a sheath owing to the reaction of the host cell, with the result that both haustorium and cell are killed. Other host cells which are penetrated, behave in the same way, so that ultimately the infection hypha dies, surrounded by a nest of dead host cells. The small yellow flecks often seen on the leaves of immune and resistant varieties are groups of dead or moribund cells, which have been violently attacked by the parasite.

As Biffen [17] first showed in regard to Yellow Rust (*v.* p. 245), susceptibility to or immunity from Black Rust is dependent upon one or more Mendelian factors. Melchers and Parker [18] and Aamodt [19] have shown that in hybrids between two varieties of common wheat, one immune from and the other susceptible to *P. graminis*, immunity is a clean cut dominant, so that in the F_2 generation there is a proportion of three immune plants to one susceptible. Puttick [20] found that in a cross between a common and a 'durum' wheat there was evidence

of the existence of more than one factor in the determination of immunity from or susceptibility to *P. graminis*.

In countries where *P. graminis* is a menace to wheat growing, much work is being done by hybridization to build up types of bread wheats which will be resistant to the fungus, notably in East Africa*. Some progress has already been achieved along these lines. In N. America the matter is complicated because of the existence of so many biologic forms, but there is hope that in time commercial varieties will be produced which will be resistant to a large number of these forms. One variety of red spring wheat, 'Webster', has been found by Stakman and others [21] to be resistant to no less than 19 forms of *P. graminis*, and, although this variety is of little value commercially, in combination with other types it may be the means of providing valuable hard red spring wheats resistant to the fungus. Another character in the wheat plant of great value in minimizing attack by *P. graminis* is that of early maturation, because if the fungus appears only when the straw is nearly ripe, little harm is done by it. Early-maturing wheats are therefore one of the great desiderata of the plant breeder in his struggle against this fungus.

Where the intervention of the barberry is necessary for the continued life of the fungus season after season the bushes should be destroyed whenever they are near arable land. In Minnesota wild barberry bushes have been killed extensively by the addition of common salt or paraffin oil to the soil.

Puccinia Phlei-pratensis. Erikss. and Henn. Timothy Rust.

This rust was separated from *P. graminis* by Eriksson and Henning [3] on the ground that it is usually incapable of infecting the barberry. It affects the stems and leaves of *Phleum pratense* and *Festuca elatior*.

Puccinia triticina, Erikss. Brown Rust of Wheat.

Aecidia on *Thalictrum* spp.

Uredospore pustules chiefly epiphyllous, 1–2 mm. long, scattered, brown; uredospores more or less globose, shortly echinulate, brownish, 16–28 μ, with 7–10 scattered germ pores.

* See W. J. Dowson, 'Wheat in East Africa', Bull. No. 4, Dep. Agric., British East Africa (Kenya Colony), 1919.

Teleutospore pustules on the leaves or sheaths, scattered, black; teleutospores oblong to clavate, truncate, slightly constricted, narrowed downwards, smooth, brown, $35-36 \times 12-23 \mu$; pedicels short; paraphyses numerous, brownish, more or less curved, on the margin of the pustule.

The heteroecious nature of this wheat rust has been determined in the United States, where Jackson and Mains [22] have shown that the aecidia occur on several species of Thalictrum. In England, however, the aecidial stage probably does not occur, and, as Mehta [9] has indicated, the fungus survives from season to season by means of uredospores. The latter retain their vitality at a lower temperature than do the uredospores of *P. graminis*. At low temperatures there may be a prolonged incubation period between infection and the formation of new pustules. Although during mild winters in England the uredospores may be found sparsely, this species does not usually become conspicuous until late in the season, frequently not until a few weeks before harvest. In consequence, Brown Rust does little harm in this country, although in other countries, as in the Argentine, where it appears earlier in the growth of the crop, considerable damage may be done by it. A Chinese variety of wheat is very resistant to *P. triticina*, and this is being used by plant-breeders in building up new commercial varieties resistant to it.

The uredo pustules of this fungus, which are formed chiefly on the leaves, are slightly lighter brown in colour and are smaller than those of *P. graminis*. Under English conditions teleutospores are comparatively rare. Several distinct biologic forms of this rust occur in N. America.

Puccinia anomala, Rostrup (= *P. simplex*, Erikss. and Henn.) Brown Rust of Barley.

The uredo- and teleutospores of this rust are essentiallly the same as in *P. triticina*, but the brown uredospore pustules are usually very small. The teleutospores germinate in spring, and it has been shown by Tranzschel [23] in Russia that the aecidia of this rust are produced on *Ornithogalum umbellatum*.

The Brown or Dwarf Rust of barley commonly occurs in England and in other countries, but it is of little economic

importance. The uredo pustules occur chiefly on the leaves. The aecidia have not been found in England, and it is probable that the fungus survives the winter in this country in the uredospore condition.

Waterhouse [23a] has shown in Australia that in certain hybrids between susceptible and resistant varieties of barley the inheritance of resistance depends on a single dominant factor.

Puccinia secalina, Grove (= *P. dispersa* Erikss.) Brown
    Rust of Rye.

The uredospore and teleutospore pustules are similar to those of *P. triticina,* as are also the spores. The aecidial hosts are *Anchusa arvensis* and *A. officinalis.*

This fungus is rigidly specialized to rye, on the leaves of which uredospore pustules are common late in the growth of the plants. The teleutospores germinate as soon as they are mature, the sporidia infecting species of Anchusa. This rust is of little economic importance.

Puccinia bromina, Erikss. (= *P. Symphyti-bromorum,* F.
    Müll.) Brown Rust of Brome Grasses.

The uredospore and teleutospore pustules are similar to those of *P. triticina,* but the aecidial host is *Symphytum officinale.*

This brown rust is of common occurrence on brome grasses. It is of particular interest because of the work of Marshall Ward [24, 25] and others upon its biologic forms. In general, he found that separate biologic forms existed on the different groups of species into which the genus may be divided, and that the fungus did not readily pass from one group of species to another. Thus spores from *Bromus sterilis* would not generally infect *B. secalinus,* and reciprocally. He concluded, however, that *B. Arduennensis* and its variety *villosus* enabled the fungus to pass from *B. sterilis* to *B. secalinus,* and that *B. Arduennensis* and its variety *villosus* were ' bridging hosts '. In view, however, of the lack of evidence of the existence of ' bridging host ' species in connexion with other rust fungi, it is desirable that Marshall Ward's work should be repeated.

Puccinia glumarum, (Schm.) Erikss. and Henn. Yellow
       Rust of Wheat and other Cereals.

Uredospore pustules on the leaves, stems, and inflorescence, about
1 mm. long, often arranged in long lines, lemon-yellow ; uredo-
spores more or less globose, echinulate, yellow, with a colourless
membrane, 25–30 × 18–26 μ.

Teleutospore pustules arranged in fine lines, black; teleuto-
spores clavate, rounded, slightly constricted, attenuated below,
smooth, brown, 30–70 × 12–24 μ ; pedicels very short or almost
none ; paraphyses brown, curved, surrounding each group of
teleutospores.
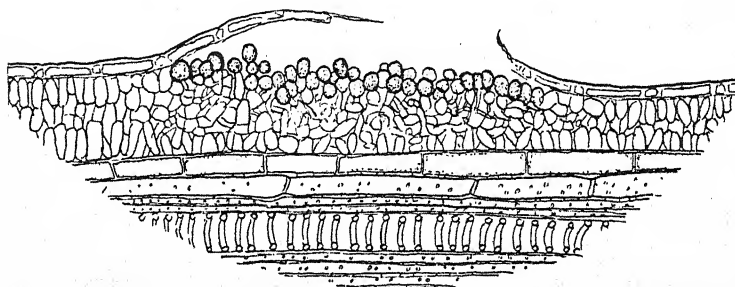
FIG. 43. *Puccinia glumarum* : section through uredo pustule, × 200.
(*R. W. Marsh.*)

This rust occurs on wheat, barley, rye, and on several grasses,
including *Dactylis glomerata* and *Agropyrum repens*. In
England it is commonest on wheat. The sporidia from the
germinating teleutospores will not infect the same hosts, and
no aecidial stage is yet known ; possibly the fungus is no
longer heteroecious.   In England Yellow Rust is usually to be
found in the uredo-stage on wheat throughout the year.   In
some seasons it is abundant as early as February, and it is
nearly always prevalent by May.   Mehta [9] has shown that the
uredospores withstand many degrees of frost, but are often
killed by high summer temperatures. After the very hot
summer of 1921 *P. glumarum* was absent from the Cambridge
area throughout the following autumn and winter.

Although the fungus commonly occurs on most varieties of
wheat cultivated in England, it causes only a slight reduction
in yield, amounting usually to not more than 5 to 10 per cent.

This loss is brought about by premature destruction of the foliage and failure of the grain to 'swell' properly. Some foreign varieties of wheat, however, when cultivated in England, are so seriously affected by this rust as to produce no harvestable grain.

There are considerable differences in varietal susceptibility. 'Rivet' and 'Little Joss' are usually resistant, and 'Wilhelmina', 'Squarehead's Master', and 'Yeoman' moderately susceptible. Wheat attacked by bunt is liable to be particularly severely affected by *P. glumarum*. Soil and weather conditions are also of great importance in determining the intensity of attack by yellow rust. On light soils and on shallow soils overlying chalk attack by this rust is usually less severe than on deep, loamy, and heavy soils. This difference is possibly bound up with a deeper root range and greater nitrogen-availability in the latter soils. Wheat which may be severely rusted in March may 'grow away' from it later in the season.

Marryat [26] has shown that immunity of some varieties of wheat, e.g. 'Einkorn', from yellow rust is due to a too vigorous onslaught by the fungus on its host, resulting in the death of groups of cells; this prevents the fungus from penetrating farther and establishing a quasi-symbiosis with the host. American workers refer to such hosts as being 'hypersensitive' to attacks of the parasite.

Biffen [17] was the first to show in connexion with this rust that susceptibility to a specific disease may be a definite genetical character transmitted hereditarily in accordance with Mendel's law. By hybridizing 'American Club' (very resistant) with 'Michigan Bronze' (very susceptible) he found that all the first generation hybrids were susceptible. On self-pollinating these, the plants of the second generation were found to segregate in the proportion of three susceptible plants to one very resistant plant, resistance in this instance being the recessive character. The behaviour in subsequent generations was in accordance with Mendelian expectations. In this case, therefore, susceptibility or resistance is dependent upon a single Mendelian factor. Upon this discovery

the way lay open to combine by hybridization the character
of resistance to Yellow Rust with other desirable qualities.
This has been done to an appreciable extent, and the variety
' Little Joss ' has come into commercial prominence in England
in this connexion. Armstrong [27] has since traced through
several generations the reaction to Yellow Rust of the deriva-
tives of hybrids between ' American Club ' (very resistant) and
' Wilhelmina ' (susceptible).

Yellow Rust of wheat occurs in other parts of the world,
but it is often overshadowed in importance by the Black and
Brown Rusts. *P. glumarum* is uncommon in the United
States except towards the Pacific coast.

Yellow Rust is usually infrequent on barley and rye in
England. As pointed out by Eriksson [10], the forms of Yellow
Rust on wheat, barley, and rye are biologically distinct.

Puccinia coronata, Corda   Crown Rust of Oats and Grasses.

Aecidia chiefly hypophyllous, seated on yellow or purplish, dis-
torted spots, cylindrical, with a whitish, torn, revolute margin ;
aecidiospores sub-globose, delicately verrucose, orange, $16-25 \times 12-20\,\mu$.

Uredospore pustules amphigenous, pinkish when covered by the
epidermis, then orange ; uredospores globose to obovate, echinulate,
yellow, $18-27 \times 16-24\,\mu$.

Teleutospore pustules hypophyllous or on the sheaths, black ;
teleutospores flat at the summit and crowned with 5–7 darker-
coloured teeth, smooth, brown, $35-60 \times 12-22\,\mu$; pedicels short,
rather thick.

P. coronata is a heteroecious species, the aecidia being pro-
duced on species of Ramnus, the other stages occurring on oats
and certain grasses, including *Lolium perenne* and *Alopecurus
pratensis*. The teleutospores hibernate on oat straw and grass
leaves, and bring about infection of Rhamnus in the spring.
Within *P. coronata*, Corda, Klebahn [4] has differentiated
P. coronifera, Klebh. (= *P. Lolii*, Nielsen), occurring on oats
and certain grasses (including rye grasses) and infecting
Rhamnus catharticus, from *P. coronata* in the narrow sense,
which affects grasses only and which has its aecidia on
Rhamnus frangula. Work in the United States by Dietz [28]
indicates, however, that the Crown Rust of oats will infect

other species of Rhamnus besides *R. catharticus*, so it seems preferable to retain the name *P. coronata*, Corda, for all forms of Crown Rust on oats and grasses.

This rust of oats and rye grasses commonly occurs where these crops are grown in proximity to Rhamnus bushes; if oats are affected at an early stage of development much damage may be done, and the crop may not be worth harvesting. In Britain, however, the fungus often develops late, if at all, and comparatively little harm is done by it to most varieties. It is most prevalent in the western and northern parts of the country. In the warmer parts of Britain the uredospores may possibly retain their vitality over the winter.

Eriksson[10] has distinguished several biologic forms in Sweden, that on oats being usually incapable of infecting grasses. Further research is required as to the existence of biologic forms of this rust in other countries.

Among varieties of oats cultivated in Britain there are considerable differences in susceptibility, the 'Scotch potato oat' being particularly susceptible. Davies and Jones[29] have found that when this variety is crossed with the variety 'Red Rust-proof', which is very resistant, the character of resistance appears to be a simple dominant over susceptibility.

Puccinia maydis, Beréng. Maize Rust.

This rust is abundant in most countries where maize is cultivated. It occurs also on Sorghum. Uredo- and teleutospores are produced on these hosts, and aecidia on species of Oxalis. There is no doubt, however, that the aecidial stage can be dispensed with. The fungus is only occasionally harmful to maize during periods of excessive humidity.

Phragmidium, Link

Teleutospores stalked, 2- to several-celled by cross septa, wall verrucose.

Aecidiospores formed in ill-defined caeomata, not bounded by a peridium.

Uredospores also occur.

All species are autoecious.

Phragmidium mucronatum, (Pers.) Schlecht (= *P. subcorti-cium*, Wint.)   Rose Rust.

Aecidiospores verrucose, orange yellow, 24–25 × 18-21 *μ*.

Uredospores ellipsoid or ovate, echinulate, orange yellow, 21–28 × 14–20 *μ*.

Teleutospores ellipsoid, usually of 6–8 cells, with a pointed papilla, blackish brown, 65–120 × 30–45 *μ* ; pedicels persistent, as long as the spore, swollen at the base, hyaline.

This fungus occurs both on wild and on cultivated roses. The aecidiospores appear first in the season; they may develop in small caeomata on the under surface of the leaves through infection by sporidia, or in long, sometimes irregular, caeomata formed from a perennial mycelium in the stems. Orange uredospore pustules arise later on the under surface of the leaves, and these are followed by groups of black teleutospores. The latter fall to the ground with the leaves and germinate in the following spring.

FIG. 44. Teleutospores of *Phragmidium mucronatum*, × 210. (*G. O. Searle.*)

This rust is sometimes so prevalent on cultivated varieties as to cause premature defoliation, but its virulence varies greatly with the season. It is most serious when the perennial mycelium is established in the stem ; such bushes are greatly weakened and may die back. Stems bearing the perennial mycelium should be cut out.

Phragmidium Rubi-ideai (Pers.), Karst.   Raspberry Rust.

All spore stages are produced on the leaves of the raspberry, the aecidiospores and uredospores being yellow-orange in colour, and the teleutospores black. Infection occurs in spring, when the over-wintered teleutospores germinate. The fungus is fairly common on cultivated raspberries, which are sometimes defoliated by it.

Phragmidium violaceum (Schultz.), Wint., and *P. Rubi*, Wint., commonly occur on the blackberry (*Rubus fruticosus*) ; the former occurs also on the loganberry.

Kuehneola, Magnus

The teleutospores are similar to those of Phragmidium, but the walls are light in colour and smooth, and the germ pores apical instead of chiefly lateral.

Kuehneola desmium, Berk. and Br. (= *K. Gossypii*, (Lager.) Arth.)   Cotton Rust.

The uredospore stage of this rust occurs in most countries where cotton is cultivated, but teleutospores have been found only in the United States. The fungus is of little importance, but it may cause defoliation of weakly plants.

Gymnosporangium, Hedw. fil.

Teleutospores formed in large erumpent masses, gelatinous and yellow when moist ; teleutospores 2-celled with long stalks which become gelatinous.   The teleutospores germinate directly in spring ; the mycelium of this stage is perennial.

Aecidia more or less elongated or tubular with a well-defined peridium ; aecidiospores yellowish brown.

Uredospores absent.

Nearly all species are heteroecious, the teleutospore stages occurring on the Cupressineae (especially Juniperus) and the aecidia on Rosaceae.

Gymnosporangium Juniperi-virginianae, Schw. (= *G. macropus*, Lk.).

This species occurs in N. America and alternates between the apple and the red cedar (*Juniperus virginiana*). The aecidial stage affects both the leaves and the fruits of the apple. The teleutospore mycelium is perennial in the red cedar and produces large excrescences ('cedar-apples') on the trees. Apples are most severely affected in regions of high humidity where the alternate host is in proximity. Attempts have been made to control the disease on the apple by spraying, but with only partial success. Where possible, diseased junipers should be eradicated.

Other species of Gymnosporangium occur on the apple in N. America.

Gymnosporangium tremelloides, R. Hertwig (= *G. juniperinum*, (L.) Mart. = *G. penicillatum*, Liro)

This is a European species alternating between the apple and species of juniper (including *J. communis*). It sometimes causes

premature defoliation of apple-trees in Norway. The life-history is similar to that of the preceding species.

Gymnosporangium sabinae, Wint.

This is also a European species which alternates between the pear and various species of juniper, including *J. sabinae.*

Gymnosporangium confusum, Plowr.

The aecidia occur on the medlar, quince, and hawthorn, and teleutospores on *Juniperus sabinae.*
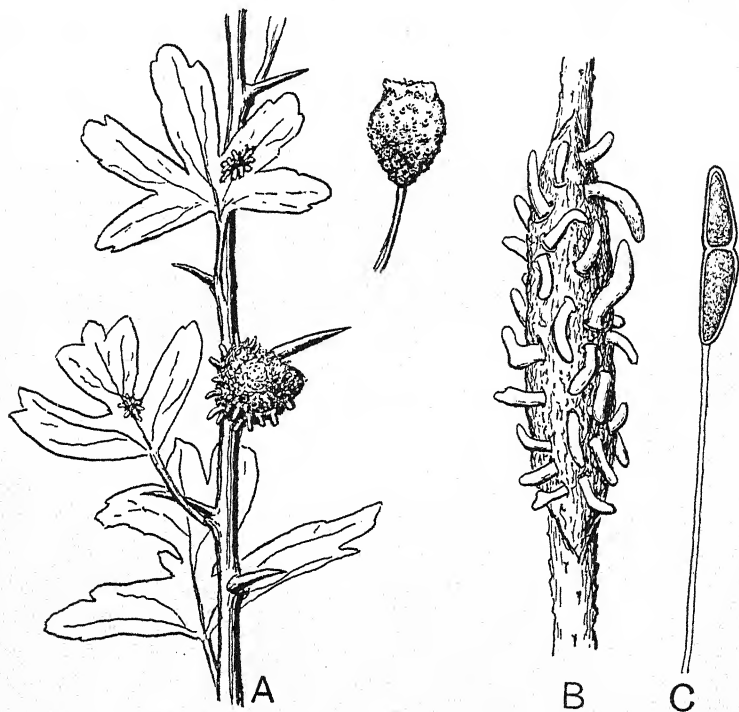
Fig. 45. *Gymnosporangium clavariaeforme* : (A) aecidia on hawthorn, natural size ; (B) teleutospore masses protruding from swollen stem of *Juniperus communis*, natural size ; (c) teleutospore, × 350. (*R. W. Marsh.*)

Gymnosporangium clavariaeforme, (Jacq.) Rees

This species alternates between the hawthorn and the common juniper. It occurs commonly in Europe, including Britain.

Cronartium, Fries

Teleutospores unicellular, formed in narrow, columnar masses which are horny when dry, germinating as soon as mature.

Aecidia yellow, with a broad, inflated, irregularly torn peridium.

Uredospores also occur.
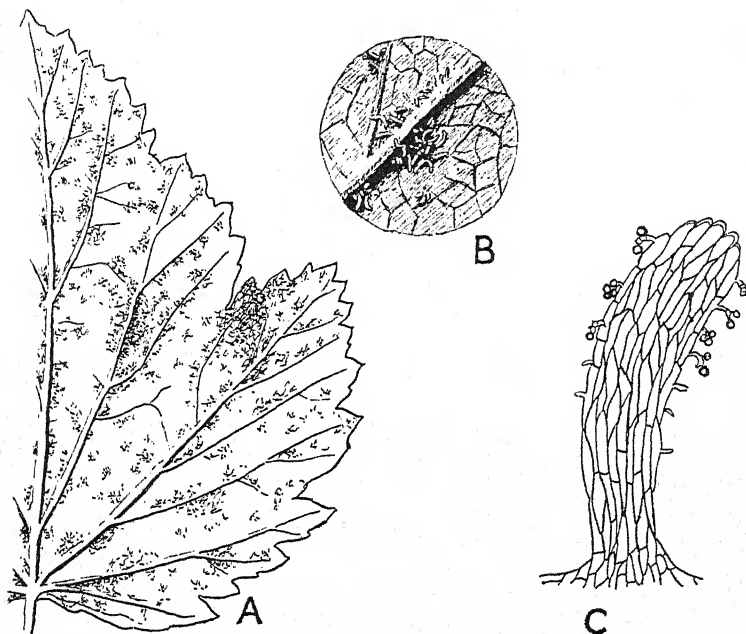
FIG. 46. *Cronartium ribicola* : (A) under surface of black currant leaf with columns of teleutospores, natural size ; (B) part of the same, × 4 ; (c) column of teleutospores with sporidia (basidiospores), × 350. (*R. W. Marsh.*)

Cronartium ribicola, Dietr. Blister Rust of Five-needled Pines; Currant Rust.

Aecidia large, about 1 cm. across, with an inflated peridium, erumpent from the bark, yellowish-white ; aecidiospores coarsely verrucose, orange, $22-29 \times 18-20 \mu$.

Uredospores echinulate, brownish-yellow, $21-24 \times 14-18 \mu$.

Teleutospores formed in columns about 2 mm. long, oblong, brownish, $70 \times 21 \mu$.

The aecidial stage of this fungus is confined to five-needled pines, in the stems of which the mycelium is perennial ; uredo-

and teleutospores are produced on the leaves of various species of currant, especially the black currant.

The fungus is a particularly dangerous parasite on young five-needled pines, as, owing to the perennial nature of the mycelium, it cripples their growth from the standpoint of the production of timber, especially in the case of the Weymouth pine (*P. strobus*). Large inflated aecidia are produced each spring on diseased stems, the spores from which infect currant bushes. The mycelium in the pine stems may produce cankers, which may lead to death of the upper part of the tree. The fungus has usually no severe effect upon currant bushes, although it may cause premature defoliation. The hair-like columns of teleutospores may almost cover the entire under surface of the leaf. The sporidia from the germinating teleuto-spores infect the pine needles, according to Clinton and McCormick [30], by way of the stomata, whence the mycelium passes into the stem. The relation between the fungus on the pine and on the currant appears to be obligate.

C. ribicola is indigenous in Central Europe, but it is now widespread in other parts of the world. Owing to the intro-duction of diseased nursery stock, as indicated by Spaulding [31], it has caused great damage in the natural forests of the Wey-mouth pine in the United States. Attempts have been made in that country to prevent the spread of the fungus by the eradication of currant bushes from the immediate vicinity of five-needled pines, but with only partial success.

In Scotland this rust on the Weymouth pine is sometimes prevented from sporing owing to attack by another fungus parasite, *Tuberculina maxima*.

Cronartium asclepiadeum, (Willd.) Fr.

This fungus attacks the stems of the Scots pine in the same way as *C. ribicola* affects five-needled pines, and the aecidia are similar. The uredo- and teleutospore hosts in France and Switzerland are species of Paeonia, Vincetoxicum, Nemesia, &c.

Cronartium Peridermium-Pini, (Willd.) Liro

This species is similar to the preceding one in producing blister-like aecidia in the bark of the Scots pine, but the alternate hosts are *Pedicularis palustris* and *P. sceptrum carolinum*.

Peridermium Pini, (Willd.) Klebh.

This is the name given to a rust fungus which occurs only in
the aecidial stage on the stems of the Scots pine, the aecidia
being large and produced by a perennial mycelium. Klebahn [32]
has shown that in northern Germany the aecidiospores infect
other pines directly, leading to the formation of aecidia again.

In the British Isles there also occurs a rust fungus, with
perennial mycelium and large aecidia, on the stems of Scots
pines, but its life-cycle has not yet been investigated. It may
be either *P. Pini,* (Willd.) Klebh., or *Cronartium Peridermium-
Pini,* (Willd.) Liro. Pethybridge [32a] has pointed out that this
' Bladder Rust' has a very serious effect on the growth of the
trees and may even kill them.

Chrysomyxa, Unger

Teleutospores forming pulvinate pustules, one-celled, germinating
directly.

Uredospores and aecidiospores occur in some species.

Chrysomyxa Abietis, Unger   Spruce Leaf Rust.

Teleutospores only occur in this species. These are formed in
golden yellow pustules on the leaves of the spruce (*Picea
excelsa*) in the spring. The sporidia infect other spruce needles
directly. The fungus was unknown in Britain before 1900,
but since then it has become common in the northern part of
the country. The fungus does not usually do much damage,
but it may cause partial defoliation.

Chrysomyxa Rhododendri, de Bary

This is a heteroecious species, alternating between the spruce, on
the leaves of which aecidia are formed, and *Rhododendron hirsutum*
and *R. ferrugineum.*

Melampsora, Cast.

Teleutospores unicellular with a brown membrane, compacted into
flat, dark coloured crusts.

Uredospores intermixed with capitate paraphyses.

Aecidia of the caeoma type. Most species are heteroecious.

Melampsora Allii-Salicis-albae, Klebh.

There are a large number of species of Melampsora on willows, the life-histories of which have been worked out by Klebahn[4]. In this particular species the aecidial hosts are *Allium ursinum* and other species of Allium, the alternate host being *Salix alba*.

The life-history of this species is peculiar in that uredospores are produced in large pustules on the stems from a mycelium which is perhaps perennial, as well as in small pustules on the leaves. The mycelium in the stems is detrimental to one form of osier (*S. alba*, var. *vitellina*) as it renders the rods too brittle for basket making. The teleutospores do not germinate until the following spring, when Allium leaves become infected.

The species of Melampsora which occur on other kinds of osier willows in Britain and which often cause considerable loss require investigation.

Melampsora Larici-Tremulae, Klebh.

The uredo- and teleutospores occur on the leaves of *Populus tremula* and other species of poplar, and the aecidia on the needles of the larch.

Melampsora pinitorqua, Rostr.

This fungus alternates between poplars (including *P. tremula*) and the Scots pine. The sporidia infect young shoots of pines under 13 years of age, producing a perennial mycelium which either kills the shoots or causes them to become twisted. This mycelium gives rise to aecidia. In view of the relationship between the alternate hosts, aspen poplars should not be allowed to grow near pine nurseries.

Melampsora Rostrupii, Wagner

This species produces its uredo- and teleutospores on the leaves of poplars (including *Populus tremula* and *P. alba*), and aecidia on *Mercurialis perennis*. The confluent and widely effused aecidia are conspicuous in the spring on the leaves and stems of the latter host.

Melampsora Allii-populina, Klebh., and *Melampsora Larici-populina*, Klebh.

Both species have uredo- and teleutospores on *Populus nigra*, *P. balsamifera*, and other poplars. The aecidia of the former rust occur on species of Allium, those of the latter on larch needles. *M. Larici-populina* is sometimes so severe on poplars in Britain as to check their growth through causing successive premature defoliations.

Melampsora Lini, (Pers.) Desm.   Flax Rust.

The orange aecidia appear first on the leaves of young plants. Uredospore pustules are then formed on the leaves, stems, and flowers, and lastly the brownish-black groups of teleutospores appear on the stems, leaves, and bolls. The teleutospores germinate in the following spring and bring about infection again. This disease of flax has been investigated in Ireland by Pethybridge and Lafferty [33] and by Pethybridge, Lafferty, and Rhynehart [34]. The brownish-black areas on the stems produced by the teleutospores are spoken of as 'firing' of flax. The presence of the rust on the stems not only spoils their appearance but renders them liable to break, and it may seriously affect the fibres. The disease is sometimes transmitted with the seed, through the inclusion of fragments of bolls and branches bearing teleutospores. Some varieties of flax grown for linseed are immune from or resistant to this rust, and in the United States such varieties are now largely grown. Henry [35] has found that if a susceptible variety is crossed with an immune one, the F_1 hybrids are immune. In the F_2 generation of one of these hybrids segregation occurred in the proportion of 15 immune plants to 1 susceptible.

Another biological form of this rust occurs on *Linum catharticum*, but spores from this species cannot infect cultivated flax.

Melampsoridium, Klebahn

This genus differs from Melampsora in that the uredospores are not accompanied by paraphyses and the aecidia are bounded by a well-defined, inflated peridium.

Melampsoridium betulinum, Klebh.

This heteroecious species alternates between the common birch and the larch. The teleutospores remain dormant in the fallen birch leaves during the winter and germinate in the spring, when the larch needles become infected. The fungus is often common on birches where the aecidial stage does not occur, and in such cases the mode of perennation is uncertain.

Melampsorella, Schröter

The genus differs from Melampsora in that the teleutospores have a thin, colourless membrane and germinate at once. The aecidia have a well-defined peridium.

Melampsorella caryophyllacearum, Schröter

The aecidia occur on the leaves of *Abies pectinata,* and the uredo- and teleutospores on the leaves of *Cerastium arvense, Stellaria media,* and closely related species. The aecidial mycelium is perennial in the branches of the silver fir and produces knot-like or cankerous swellings, from which 'witches brooms' arise. The latter consist of a number of small, erect branches on the leaves of which the aecidia are formed in summer. The 'witches broom' is deciduous, the leaves being smaller and rounder than normal. Some of the 'witches brooms' on silver firs are not caused by this parasite, but are of the nature of hereditary monstrosities.

Coleosporium, Lév.

Teleutospores formed in waxy pustules, orange-red, with a gelatinous membrane. The teleutospore becomes a basidium, from each cell of which a sporidium is formed directly at the end of a long sterigma.

Uredospores formed in chains.

Aecidia with an inflated peridium, which becomes irregularly torn.

Coleosporium Senecionis, Fr.

The orange pustules of uredospores and the red, crust-like groups of teleutospores are formed on the under surface of the leaves of species of Senecio, including *S. vulgaris, S. jacobaea,*

and *S. silvaticus,* and on Cineraria. The aecidia occur as yellowish-white blisters on the needles of the Scots pine in spring, which have been infected by sporidia in the previous autumn. This stage was formerly named *Perider-mium Pini,* var. *acicola.* The fungus is frequently seen on pines in the neighbourhood of infected Senecios. The fungus is not harmful to adult pines, but the alternate host should be eradicated from pine nurseries. The rust occurs commonly on groundsel plants remote from pine trees, and it seems likely that the fungus can over-winter in some parts of England in the uredospore condition.

FIG. 47. Aecidia of *Coleosporium Senecionis,* natural size. (*R. W. Marsh.*)

The following species of Coleosporium also produce similar aecidia on the needles of the Scots pine :

C. Tussilaginis, Tul., the alternate host being *Tussilago farfara.*

C. Petasites, Lév., the alternate host being *Petasites officinalis.*

C. Sonchi, Lév., the alternate hosts being species of Sonchus.

C. Campanulae, Lév., the alternate hosts being species of Campanula. This species probably overwinters in the uredospore condition.

Endophyllum, Lév.

FIG. 48. Germinating teleutospores of *Endophyllum Sempervivi,* × 350. (*W. J. Dowson.*)

In this genus teleutospores only are formed, which arise in chains after the manner of aecidiospores in other genera.

Endophyllum Sempervivi, de Bary

This rust occurs on wild and cultivated Sempervivums. The sporidia infect the leaves, from which the mycelium passes into the root-stock, where it is perennial. The mycelium causes

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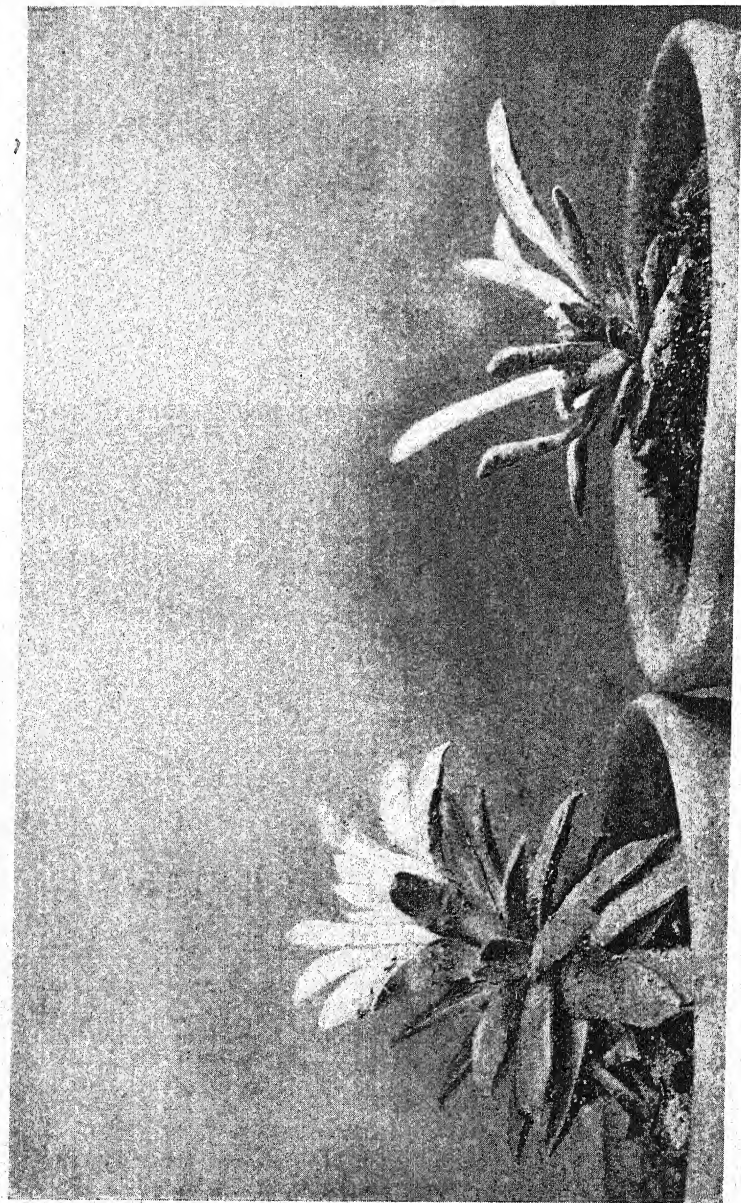
Fig. 49. Houseleeks (*Sempervivum* sp.) affected by *Endophyllum Sempervivi*, × 2. (*W. J. Dowson.*)

the rosettes to be less compact than usual, and the infected leaves are more erect and longer than healthy ones. The teleutospores are produced in small, orange-coloured cups on the leaves, which are often accompanied by spermogonia.

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FUNGUS DISEASES (*continued*): AURICULARIALES, EXOBASIDIALES, APHYLLOPHORALES

AURICULARIALES

BASIDIA transversely septate, arranged in a definite hymenium which is exposed from the first. Each cell of the basidium produces one spore at the end of a long sterigma. Fruit-bodies usually large.

Auricularia, Bull.

Fruit-body gelatinous when moist, cartilaginous when dry, dimidiate or cup-shaped, the lower surface forming the hymenium.

Auricularia auricula-Judae, (L.) Schroet. Jews' Ear Fungus.

This fungus is a slow-growing parasite on old elder trees, and more rarely on beech and elm. Infection occurs through wounds, and the mycelium spreads chiefly in the wood, delignifying it. As the branches die back, the fructifications appear. These are 2–8 cm. across, ear-shaped, and brownish in colour; they are soft and gelatinous when moist, but horny when dry. The fungus also grows as a saprophyte.

EXOBASIDIALES

Basidia non-septate, arranged in an ill-defined hymenium on leaves, &c., in which the mycelium is parasitic. The spores are formed on sterigmata at the apex of the basidium.

Exobasidium, Woronin

Hymenium discontinuous; basidia cylindrical, with 4–6 sterigmata; mycelium parasitic in leaves and stems, causing hypertrophy. Conidia may be formed between the basidia.

Exobasidium Vaccinii, (Fuckel) Wor.

This fungus produces galls on the leaves and flowers of wild Rhododendrons and on Azaleas cultivated in greenhouses. The galls on the leaves vary in size from that of a pea to a small plum; they are sometimes irregular in shape. The galls

are reddish when young, but become white when mature owing to the formation of a delicate bloom of spores. The parasite is sometimes troublesome in the cultivation of Azaleas. The galls should be cut off before they produce spores, and insects should be kept under control by spraying or fumigating, as these disseminate the spores. The complete life-history of the fungus is still somewhat obscure.

Exobasidium vexans, Massee   Blister Blight of Tea.

'Blister Blight' of tea is known at present only in northern India and in Formosa. The galls or blisters on the tea leaves are reddish in colour and a quarter to half an inch in diameter. Many blisters may occur on a single leaf; only young leaves can be infected. The under surface of the blister ultimately becomes white owing to the formation of spores. After the leaves have been attacked, the fungus spreads to the leaf stalks and young green stems. Spots produced on these stems extend until the stems are girdled, when the upper part dies and breaks off. In this way the fungus is sometimes very harmful. The disease breaks out first on unpruned tea bushes, whence it spreads to the young shoots of pruned tea. Most damage is done where the atmosphere is very humid. The disease can be controlled by spraying the bushes with Bordeaux or Burgundy mixture just before the rains commence and during the early part of the wet season. A solution of 2 oz. of salt and 2 oz. of lime in one gallon of water has also been found to be a useful preventive spray of 'Blister Blight' in wet weather. The expense of spraying is heavy, but is warranted with new plantations and with bushes kept for seed. Where the disease appears on small areas it is advisable to pluck off the affected leaves and bury them on the spot.

APHYLLOPHORALES

Basidia non-septate, spores (usually 4) formed at the apex, at the extremity of slender sterigmata. Basidia, usually interspersed with paraphyses, arranged in a definite hymenium, which is exposed throughout development and which is spread over pores, teeth, anastomosing gills or a smooth surface. The fruit-bodies are usually large and of various consistencies. Clamp connexions are

frequently present in association with the septa of the vegetative mycelium. This group of fungi is so large that the characters of the families of pathological importance are indicated.

CLAVARIACEAE

Fruit-body erect, simple or branched, usually fleshy, the upper part bearing the hymenium all round it.

Typhula, (Pers.) Fr.

Fruit-body often growing from a sclerotium, with a long, thin stem, generally unbranched; basidiospores white in mass.

Typhula graminum, Karst.

Young cereal plants on the Continent are sometimes killed by this fungus in the spring. The sclerotia, which are reddish-brown in colour and 1–2 mm. across, develop on the stems and leaves near soil level and serve as a means of dissemination. On germination in the following spring the sclerotium produces a slender fruit-body which forms basidiospores.

Other species of *Typhula* occasionally attack other crops. *T. Betae*, Rostr., affects sugar beet, and *T. Trifolii*, clover.

The minute sclerotia of these species are sometimes distributed with the seed.

THELEPHORACEAE

Fruit-body resupinate or bracket-like; hymenium spread over a smooth, rugose or ribbed surface.

Corticium, Persoon

Fruit-body waxy, crustaceous or floccose, resupinate, effused; hymenium smooth or tubercular, continuous, often cracked; spores usually white in mass.

Corticium Solani, Bourd. and Galz.

Fruit-body thin, effused, arachnoid, pale buff colour, basidia not forming a compact hymenium; basidiospores elliptic-oblong, hyaline, $8–14 \times 4–6\,\mu$. Small, irregularly shaped, brownish-black sclerotia (*Rhizoctonia Solani*, Kühn) are frequently formed.

Many slightly differing forms of this fungus attack a great number of cultivated plants, including cotton, beet, lettuce,

carnation, potato, and tomato.   Lawn grasses may also be killed
by it.   On many of the hosts attacked by the various strains
of this fungus the basidial stage has not been seen; in such
cases the fungus persists and may be distributed by means of
sclerotia, which pass into the soil or remain adherent to parts
of the plant.   The mycelium is characterized by the lateral
branches being constricted at the point of origin.   Some forms
of the fungus can be transferred readily from one host to
another, but in others, according to Matsumato [1], there is a
considerable degree of specialization.   Several forms of the
fungus have been studied by Duggar [2] and by Briton-Jones [3].

This parasite often causes the damping off of seedling plants,
especially cotton ('Sore-Shin' disease), as described by Balls [4],
who studied its temperature relations.   He found that growth
of the fungus was inhibited at a temperature of 35° C., at
which the cotton plant readily produces a cork barrier that
prevents further growth of the fungus even if the temperature
is subsequently reduced.   The hyphae formed by the sclerotia
on germination penetrate the roots or stems at about soil level,
and cause a soft rot.   The fungus may also attack the host
from the soil at a later stage of development, as with tomatoes
under glass.   With this crop Small [5] states that the fungus is
most virulent at a temperature of 16–20° C. and in soil of
high water-content.

The sclerotia are often seen as black blotches on the surface
of potato tubers ('Black Scurf'), but they are superficial and
harmless to the tubers.   The sclerotia give rise to active
mycelia when the tubers are planted, and these hyphae may
invade and kill the young shoots.   The mycelium also some-
times kills the young shoots before planting if the seed tubers
are 'sprouted' in a humid atmosphere.   Often, however, under
English conditions the mycelium is relatively innocuous to the
growing potato plant, and it ultimately forms a greyish film
around the base of the stems, which bears the hymenium, but
which remains entirely superficial.   According to Dana [6] the
roots of potato plants in the State of Washington may be so
injured by this fungus as to cause a serious reduction in
yield.

In tomato cultivation under glass the most effective preventive of this disease is soil sterilization by steam. The sclerotia on potato tubers can be killed by immersion in weak corrosive sublimate or formalin, but it is not usually worth while to treat the tubers. Where the disease is harmful in potato cultivation only tubers free from sclerotia should be planted. The disease usually damages the potato crop only where potatoes are grown on the same land year after year.

Corticium fuciforme, (Berk.) Wakef.

Grass in lawns and pastures is occasionally killed by this fungus, formerly known under the name of *Isaria fuciformis*, the pink mycelium of which spreads over the turf. The fruit-bodies are small, incrusting, and rose-coloured.

Corticium salmonicolor, Berk. and Br.   Pink Disease.

Fruit-body thin, effused, rose-pink but rapidly fading, becoming cracked; basidia tetrasporous; basidiospores pyriform, apiculate, 9–12×6–$7\,\mu$.

Conidial pustules ($= Necator\ decretus$, Mass.) orange-red; conidia irregularly oval or spherical, hyaline, 14–20×8–$10\,\mu$.

'Pink Disease' of tropical plants is widely distributed throughout the eastern and western tropics, and may cause considerable losses in rubber, tea, coffee, cocoa, cinchona, and other plantations. The fungus is omnivorous and has been recorded as a parasite on more than 100 different hosts. Pink Disease has been investigated by Petch[7], Rant[8], Brooks and Sharples[9], and others.

The appearance of the fungus and the symptoms induced by it are extremely variable. The mycelium is at first entirely superficial and cobweb-like on the stem, but it gradually becomes transformed into a series of pinkish pustules or a more or less continuous pink incrustation. Some of the mycelium penetrates the bark and may enter the wood, often killing the upper part of the tree or bush. On the other hand, the growth of the mycelium in the bark may be checked, especially in dry weather, when a canker is formed. The pink incrustation on the shaded surface of the bark sometimes forms basidia, but is often sterile. Another sporing stage is formed by the aggregation of mycelium just under

the surface of the bark, especially on the side exposed to bright light, and this, becoming erumpent and separating into the constituent cells, forms an orange-red spore pustule. This stage was formerly thought to be a separate fungus and was named *Necator decretus*. In Malaya spores of this type are produced more commonly on rubber trees than are basidio-spores.

Pink disease is indigenous on jungle and other trees in the tropics, whence the spores spread to cultivated plants such as rubber, tea, &c. It is most serious in regions of high rainfall.

In the rubber plantations of the East this disease is con-trolled by cutting out and burning the affected branches if the mycelium has already penetrated the tissues. Where the fungus is still only superficial on the bark it can be checked effectively by covering the bark with tar. Opera-tions of this kind on large estates are best carried out by a 'pest gang' trained particularly to treat disease. In tea cultivation, spraying with Bordeaux mixture after the bushes have been pruned has been found helpful in controlling the disease.

Corticium koleroga, (Cke.) v. Hohn.   (= *Pellicularia koleroga*, Cke.)

This fungus attacks the shoots of coffee bushes in Asia and America, covering the under surface of the leaves and the berries with a delicate film. In severe attacks leaves and fruits may be bound together by the film. The mycelium is entirely superficial, but it has a severe effect on the bushes, causing the leaves and berries to turn black. The disease can sometimes be prevented by spraying with Bordeaux mixture.

Stereum, (Pers.) Massee

Fruit-body coriaceous, resupinate or bracket-like; hymenium smooth; spores white in mass.

Stereum hirsutum, (Willd.) Fr.

Fruit-body 2–10 cm., yellowish with a yellow margin, resu-pinate or bracket-like, upper surface hairy, sub-zoned, hymenium

ochraceous, sometimes becoming grey, smooth; spores elliptical, incurved, hyaline, $6-8 \times 3-4 \mu$.

This species is usually saprophytic, occurring on oak branches, stumps, and posts, and occasionally on dead woody parts of other broad-leaved trees. It causes rapid delignification of woody tissues, as described by Marshall Ward[10]. According to Hartig[11] the fungus sometimes invades, as a wound parasite, the wood of standing oak trees, causing the appearance therein of yellow or white stripes. Such wood is said to be 'yellow piped', or 'white piped'. On the Continent this fungus, or a variety of it (*necator*), is one of the causes of vine 'apoplexy', in which branches of vines over ten years of age die back apparently suddenly, although the fungus has been present in them for some time. Infection takes place through pruning wounds. This trouble is greatly lessened by treating the wounds with sodium arsenite from time to time.

Stereum purpureum, Pers. Silver-leaf Disease.

Fruit-body 2–8 cm., brown or brownish-purple, fading with age, resupinate or more or less imbricate, upper surface hairy; hymenium lilac or purplish, becoming faded; spores oval, apiculate at one end, hyaline, $6-8 \times 3-4 \mu$. One of the characteristic histological features of this fungus is the presence of vesiculose, sub-hymenial cystidia, $15-30 \times 12-25 \mu$.

Silver-leaf disease in fruit trees and other arborescent plants is almost always due to this destructive fungus, which also occurs commonly as a saprophyte on the stumps of broad-leaved trees (especially poplar, willow, birch, elm, and beech) and occasionally on conifers. When behaving as a parasite fructifications of the fungus arise in wet weather on the branches and trunk as these die back. The fruit-bodies are purplish when actively growing, but they become dingy with age; they are extremely polymorphic, being frequently resupinate and widely effused on the under surface of lateral branches, and often bracket-like and densely imbricate on trunks and erect branches. Spores are formed by the hymenium only when the fructifications are moist, but after drying
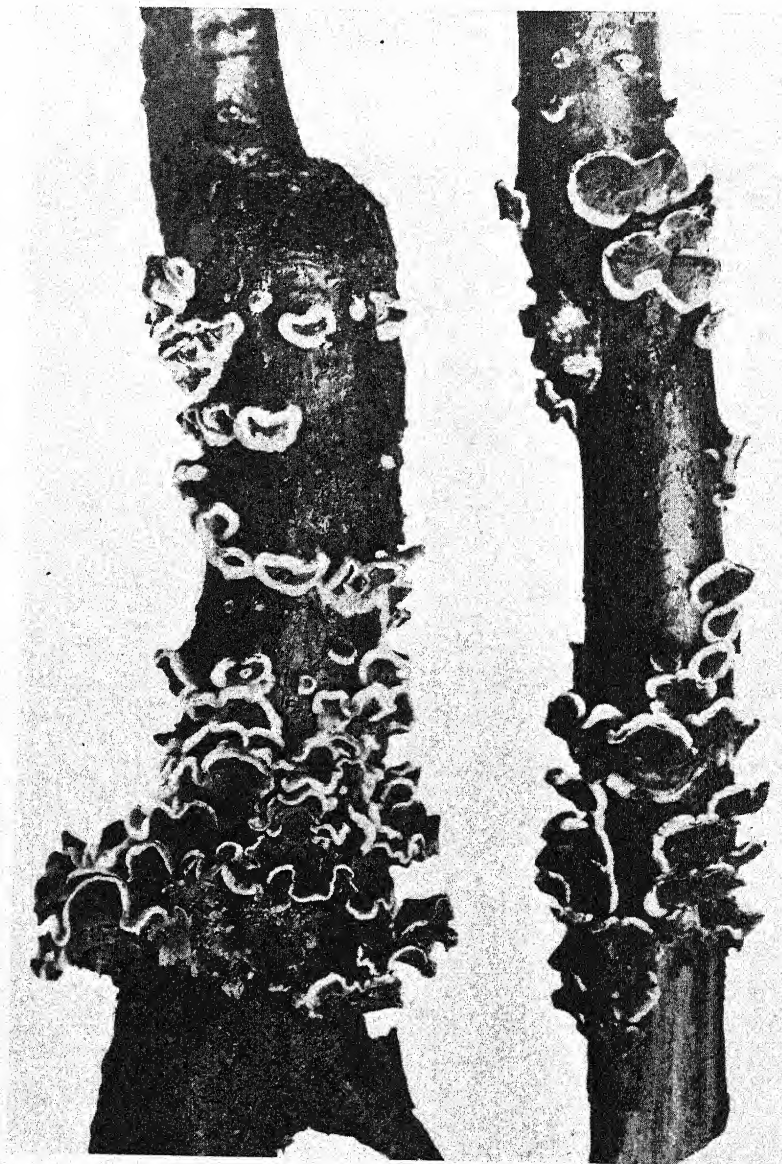
Fig. 50. Fructifications of *Stereum purpureum* on plum trees, natural size.

up the fructifications may again give rise to spores when moistened.

Silver-leaf disease of woody plants is widely distributed. It is very destructive to plum and apple trees in Britain, in New Zealand as described by Cunningham[12], and in parts of N. America as recorded by Güssow[13] and Heald and Boyle[14]. The disease also commonly affects the peach, morello cherry, and laburnum. It is of rarer occurrence on currant bushes and roses. The disease has been investigated by Percival[15], Pickering[16], and by Brooks and his colleagues[17-20].

'Silver-leaf' disease is so called because the foliage generally becomes silvery in appearance during the early stages of attack by the fungus in the woody parts below the affected foliage. The fungus is not present in the leaves, but, in connexion with its occurrence in the wood below, a disturbance is caused which leads to the silvery transformation of the foliage. Some substance is secreted by the mycelium, which, carried up in the transpiration current, produces directly or indirectly certain histological changes in the leaves. The silveriness of the foliage is primarily due to partial separation of the mesophyll cells from one another and from the epidermis. Owing to the presence of abnormal air spaces thus induced the quality of the light reflected from the surface is changed, and the leaves appear silvery. At a later stage the chloroplasts may become yellowish, and, in severely affected leaves, brown necrotic areas develop before leaf-fall. The author has recently induced silveriness and other toxic symptoms in plum leaves by injecting a filtered extract of the fungus into the stems, and this makes it clear that some product of the metabolism of the fungus is the cause of these symptoms.

On the other hand, attack of woody plants by this fungus is not invariably associated with silvering of the foliage. Birch and beech trees may be killed by *S. purpureum* without showing silvery symptoms, and Cotton[21] has described the same phenomenon in Rhododendrons. Whether or no the foliage of trees attacked by *S. purpureum* shows silvering depends probably upon the texture of the leaf.

S. purpureum causes infection through pruning wounds and broken branches. The fungus is so ubiquitous that its spores are commonly present in the air. Brooks and Moore[22] have shown that if the spores alight on a wound in the wood,
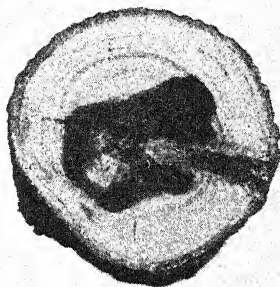
Fig. 51. Branch of a 'Victoria' plum tree, affected by Silver-leaf disease, cut across to show discoloration of the wood caused by *Stereum purpureum*. Natural size.

especially if recently made, they are frequently sucked a considerable distance into the vessels, in which they germinate under more favourable conditions than on the surface. The young mycelium spreads rapidly in the vessels, and its progress is usually accompanied by the formation of 'gum', which is produced chiefly from the carbohydrates in the wood, and which generally causes the invaded wood to become brown. The fungus spreads more rapidly in a vertical direction than laterally, but sooner or later the branches begin to die back, and the whole tree ultimately succumbs unless the growth of the fungus is checked.

Silvered trees attacked by *S. purpureum* do not, however, invariably die; they sometimes regain their health. Brooks and his colleagues[19, 20] have shown that this recovery is due to the growth of the fungus being stopped by the formation of a special 'gum barrier' around the invaded tissues, which cannot be penetrated by the mycelium. The 'gum barrier'

is formed by the reaction of the host and consists of a narrow zone of 'gum', darker in colour and more densely aggregated than that which accompanies active invasion by the fungus. The result of the formation of a 'gum barrier' is to occlude the fungus, which gradually dies out.

Natural recovery from the disease is most frequent in vigorous trees. It is particularly pronounced in varieties of fruit trees, e. g. the Pershore plum, which are usually resistant to the disease. This is associated with the rapid formation of 'gum barriers' by these varieties.

Brooks and Moore [20] have shown that newly exposed woody tissues of susceptible fruit trees can be readily invaded by the fungus throughout the year except during June, July, and August, when the reaction of the host to injury and incipient attack is so violent, even in a susceptible variety of plum such as 'Czar', as to result in the rapid formation of a 'gum barrier', which prevents infection. This difference in infectivity at different seasons is doubtless bound up with changes in the physiological state of the tree.

Woody tissues already exposed for more than a month become infected by *S. purpureum* only with great difficulty, and the longer they have been exposed the less likely are they to become infected. A fresh wound is liable to be invaded by a great variety of micro-organisms, and, unless *S. purpureum* becomes established almost immediately, it has great difficulty in penetrating tissues already occupied by other organisms and filled with 'gum' formed by them or by the traumatic response of the tissues.

Among fruit trees there are great differences in varietal susceptibility. In Britain the most susceptible varieties of plum are 'Victoria' and 'Czar', and the most resistant 'Pershore' and 'Greengage'. Among apples the most susceptible varieties are 'Newton Wonder' and 'Early Victoria'.

Silver-leaf disease has played such havoc in fruit plantations in England in recent years that the Ministry of Agriculture and Fisheries, by the terms of the Silver-leaf Order of 1923, compels fruit-growers to destroy before July 15 each year all dead woody tissues liable to harbour *S. pur-*

pureum. This has the effect of preventing the fungus from fructifying in the plantations. It is impossible to destroy completely such a common fungus as *S. purpureum*, but the risk of infection is reduced appreciably by this measure of plant sanitation. By cutting out dead branches shortly before July 15 the risk of reinfection through wounds thereby made is least, for spores of the fungus are then least abundant, and, as pointed out above, it is exceedingly difficult for the fungus to cause infection at that season. Some fruit-growers control the disease successfully in the early stages by cutting out the silvered branches early in the summer, but unless this operation is done with great care so that the lower limit reached by the fungus is excised, it is useless. Besides, silvered branches not infrequently recover, and they do not become a source of infection until dead and bearing fruit-bodies of the fungus.

Current pruning practices are a prolific source of infection by *S. purpureum.* In view of the danger from this and other destructive fungi, fruit trees should be cut about as little as possible when once the young trees have been shaped properly. Where thinning out of large branches is really necessary this should be done early in the summer, when the risks of infection are least. Broken branches should be cut off flush with the main stem or larger branch.

In order to obviate risk of infection by *S. purpureum* wounds should be covered immediately with some protective substance, preferably soft grafting wax or thick paint. A convenient paint for this purpose is made up as follows:

To 2 lb. white lead paste (as bought) add 2 teaspoonfuls of paste driers and 2 tablespoonfuls of linseed oil. Mix. Then add 2 tablespoonfuls of turpentine and mix well.

Brooks and his co-workers[19, 20] have shown that both Stockholm tar and gas tar are entirely ineffective in keeping *S. purpureum* at bay. Particular care should be taken in nurseries to protect wounds without delay.

In view of the fact that fruit trees in vigorous growth are much more likely to recover from the disease than weakly ones, every effort should be made by careful selection of

soil and stocks to ensure the maximum vigour of development. Manurial treatment of older trees, especially applications of kainit and basic slag, may be helpful in this respect on some soils.

All examples of silvered foliage are not, however, due to *S. purpureum*. Apart from 'silvering' caused by insects such as thrips, herbaceous plants frequently show silvered leaves at the end of the winter, the histological symptoms of which are similar to those induced by the fungus. Seedling plums and other plants not infrequently exhibit silvering of the leaves. Silvering of foliage is a general pathological phenomenon, which may be induced in various ways, but by far the most important from the economic standpoint is the influence of *S. purpureum* growing in the tissues of arborescent plants.

HYDNACEAE

Fruit-body fleshy, sometimes pileate, the hymenium being spread over the surface of spines, granules, or warts.

Hydnum, (Linn.) Fr.

Fruit-body usually fleshy, spines acute, distinct at the base.

Hydnum septentrionale, Fr.

This fungus causes a heart-rot of the sugar maple and other trees in North America and Europe. The fruit-bodies are sometimes more than a foot across and arise in creamy white, bracket-like clusters. The teeth are often half an inch long.

Hydnum omnivorum, Shear

According to Shear[23] this is the perfect stage of a root-attacking fungus very destructive to cotton, lucerne, and other plants in the southern United States, which was formerly known in the mycelial state as *Ozonium omnivorum* and in the conidial stage as *Phymatotrichum omnivorum*. The fungus spreads centrifugally in the soil, killing the plants as it proceeds. Taubenhaus and Killough[24] state that it needs a living host to enable it to survive the winter, so that control measures should be concentrated on the eradication of the roots of susceptible plants from contaminated soil.

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FUNGUS DISEASES (*continued*): APHYLLOPHORALES
(*continued*)

MERULIACEAE

FRUIT-BODY usually fleshy, resupinate or bracket-like, the hymenium being spread over veins or anastomosing pores, or smooth.

Coniophora, (DC.) Pers.

Fruit-body fleshy, subcoriaceous or membranaceous, resupinate, effused ; hymenium smooth or tubercular ; spores coloured.

Coniophora puteana, (Schum.) Karst. (= *Coniophora cerebella*, Pers.)

This fungus frequently causes a dry rot of worked timber in buildings under moist and badly ventilated conditions, and it sometimes precedes attack by *Merulius lacrymans*. It also occurs on stumps and felled trees. The fructifications are large, yellowish, or olivaceous, with a white margin, resupinate. The spores are olivaceous or ferruginous, and measure $11–13 \times 7–8 \mu$. Clamp connexions occur frequently in the vegetative mycelium, 3–5 of these being found near a septum.

Merulius, Fr.

Fruit-body waxy or fleshy, resupinate or bracket-like ; hymenium at first smooth, becoming reticulated with irregular obtuse folds or pores, fertile on the edge ; spores white or coloured.

Merulius lacrymans, (Wulf.) Fr.

Fruit-body 5–50 cm. across, yellow ferruginous, fleshy, resupinate or rarely partly bracket-like, margin white, folds porous, large ; spores rust-coloured in the mass, yellow under the microscope, elliptical, $8–10 \times 5–6 \mu$.

According to Falck[1] *Merulius lacrymans* comprises three distinct forms : *M. domesticus*, the common cause of dry rot in buildings, *M. minor*, the small dry-rot form, and *M. silvester*, which occurs in woodlands.

Merulius lacrymans is the commonest cause of dry rot of worked timber in buildings, and for that reason is often called the 'Dry Rot' fungus. There are, however, other fungi which cause a similar destruction of structural timber. *M. lacrymans* is also found on logs in timber yards, but is of very rare occurrence on tree stumps in the open.
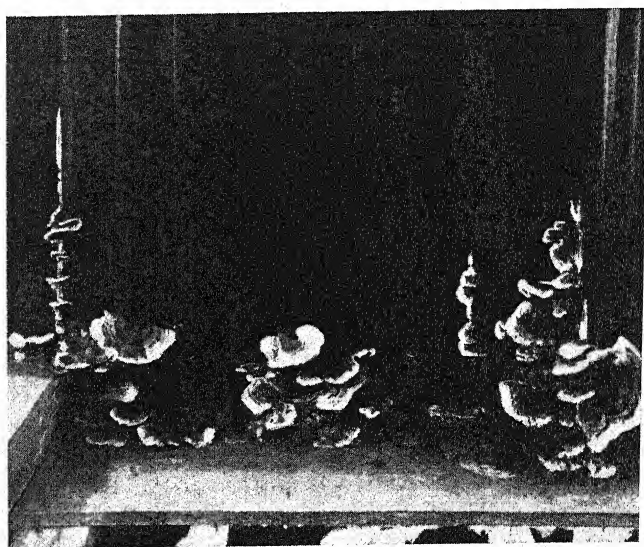
FIG. 52. Fructifications of *Merulius lacrymans* on the panelling and floor of a room ; the source of infection was under the floor, part of which has been removed. $\times \frac{1}{12}$. (*A. Smith.*)

Woodwork already attacked by *Coniophora puteana* (= *C. cerebella*) and badly-seasoned wood are especially liable to become infected by *M. lacrymans* where the ventilation is defective. The mycelium is at first superficial on the wood, but some of the hyphae penetrate and delignify the tissues, ultimately causing the wood to crumble. Much of the mycelium remains superficial and under suitable conditions hangs down in white, flocculent masses, from the extremity of which drops of water exude that give to the fungus the specific name '*lacrymans*'. The mycelium becomes ashy grey in colour with age. A characteristic feature of the mycelium is

the presence of one or two clamp connexions in connexion with some of the cross septa, these 'clamps' frequently growing out into hyphae. Amongst the aerial mycelium there often arise thick strands having considerable internal differentiation, which spread long distances over inhospitable substrata such as brickwork and cement, and by means of which woodwork in remote parts of the building may be attacked. The fructifications arise on the surface of the wood as large circular discs, although if they develop on panelling they may be more or less bracket-like, with the hymenial surface below. Sometimes the first sign of attack is the appearance on a floor of a brown deposit of spores which have drifted up through cracks from fruit-bodies below.

A detailed study of the mode of action of the fungus on wood has been made by Falck[1]. Czapek[2] has isolated an enzyme from the mycelium, which is capable of decomposing lignified membranes. A characteristic feature of wood in the last stages of attack by *M. lacrymans* is its tendency to split into quadrangular pieces. The wood of broad-leaved trees is less readily attacked by *M. lacrymans* than is coniferous timber, but even well-seasoned oak will succumb to it in time under favourable conditions.*

An enormous amount of destruction is caused by this fungus, and it is not uncommon for entire buildings to collapse in consequence of it. The fungus most frequently begins to grow in cellars and spaces under floors which are badly ventilated. Good ventilation is the greatest enemy of *M. lacrymans*, for the fungus cannot establish itself where there is a current of air. Only well-seasoned timber should be used for structural purposes, and where joists abut on walls from which water may be absorbed, they should be creosoted or treated with boric acid. If an attack of 'dry rot' is seen in an early stage it can often be effectively dealt with by improving the ventilation and removing the infected wood, and by treating the contiguous woodwork with formalin or other antiseptic.

* Further information concerning the destruction of timber by this and other fungi will be found in *The Microbiology of Celluloses, Hemicelluloses, Pectin, and Gums*, by A. C. Thaysen and H. J. Bunker, Oxford University Press, 1927.

Falck[1] states that the best substances for impregnating structural timber against attack by dry rot are soluble compounds of fluorine and arsenic.

FISTULINACEAE

Fruit-body large and fleshy, bracket-like, the hymenium lining the surface of free and separate tubes.

Fistulina, (Bull.) Fr.

Fruit-body fleshy, bracket-like ; spores coloured.

Fistulina hepatica, (Huds.) Fr.   Beef-steak Fungus.

The fruit-bodies grow on trunks of broad-leaved trees, particularly oak and ash, in which the mycelium attacks the woody tissues, producing a dark red discoloration. Affected trees are gradually killed by the fungus. The fructifications are 10–25 cm. across and dark red in colour, but the orifices of the tubes are paler. The spores are pink, $4\cdot5–5 \times 4\,\mu$.

POLYSTICTACEAE

Fruit-body bracket-like or resupinate, coriaceous or woody ; hymenium lining tubes, or covering gills or teeth, homogeneous with the substance of the pileus.

Trametes, Fr.

Fruit-body woody or corky, bracket-like or resupinate.

Trametes Pini, (Brot.) Fr.

Fruit-body large, 10–30 cm. across, perennial, bracket-like, brownish-black, rough, becoming encrusted with age ; pores yellowish-red, 6–15 mm. long ; flesh ferruginous, woody, very hard ; spores oval, pale yellow, $4–6 \times 4–5\,\mu$.

This fungus attacks the trunks of conifers more than forty years of age, being particularly prevalent on the Scots pine. Boyce[2a] states that it is the principal cause of decay of the heart wood of old Douglas Firs in N. America. The fungus usually obtains entry into the tree where a branch has broken off, but on account of the copious formation of resin in young trees it makes rapid progress only in the heartwood of old trees. Here the mycelium attacks certain

annual rings more than others, producing a 'ring shake'. At first the wood becomes darker red in colour, but later whitish blotches or holes appear. Hartig[3] and Hubert[4] have studied in detail the delignification of the wood, a characteristic feature being the early disorganization of the middle lamella. In pines and larches the fruit-bodies usually arise at places where branches have been broken off, but in the comparatively non-resinous spruce and silver-fir they appear on any part of the trunk. Trees infected by this fungus should be removed in thinning, but the timber in the lower part of the trunk is often sound.

Lenzites, Fr.

Fruit-body corky, often bracket-like; gills coriaceous, often anastomosing at the base; spores white.

Lenzites saepiaria, (Wulf.) Fr.

This fungus is one of the most important causes of the rotting of coniferous timber used in mining. It occurs also on the stumps of conifers. The fruit-bodies are about 8 cm. across, dark-brown with a yellowish margin; the gills are yellowish when young, more or less anastomosing, and very rigid.

Polystictus, Fr.

Fruit-body coriaceous, membranaceous, bracket-like, often imbricate.

Polystictus versicolor, (Linn.) Fr.

This is one of the commonest saprophytic fungi on the stumps of broad-leaved trees, causing disintegration of the wood. It may occasionally be parasitic, as described by von Schrenk[5]. The fruit-bodies are 3–8 cm. across, bracket-like, and often imbricate, the upper surface being marked with concentric, smooth, shining, satiny zones of various colours.

POLYPORACEAE

Fruit-body fleshy, coriaceous, or woody, bracket-like, stalked, sessile, or resupinate; hymenium lining tubes coherent throughout their length, forming a layer distinct from the substance of the pileus.

Poria, Fr.

Fruit-body membranaceous or corky, resupinate; tubes round or angular; spores white or coloured.

Poria vaporaria, (Pers.) Fr.

Fruit-body 5–10 cm. across, white, effused; pores white, then cream colour, 0·5–1 mm. long, large, angular; spores white, $6 \times 2\,\mu$.

This species, which is a common saprophyte on the branches of trees, often attacks coniferous timber in buildings in much the same way as does *Merulius lacrymans*. The mycelium remains persistently white, not becoming grey as in *Merulius lacrymans*. Large, irregular mycelial aggregations are often found on the surface of the wood.

Poria hypolateritia, Berk.   Red Root Disease of Tea.

Petch[6] states that this is one of the commonest causes of root disease of tea in Ceylon, especially at high altitudes. A characteristic feature of the disease is the presence on the root surface of mycelial strands which, though white and soft when young, become red and tough, and finally black with age. The rhizomorphs may spread laterally and fuse with one another, forming continuous sheets. The fructifications are produced at the collar of dead bushes, and appear as thin, flat, reddish plates studded with minute pores. The fungus usually begins to grow on old jungle stumps, whence the rhizomorphs grow through the soil to the tea roots. Care should be taken in young tea plantations to destroy affected bushes and to isolate infected soil by means of trenches.

P. hypobrunnea, Petch, also attacks tea occasionally in a similar manner, but the fructifications are slaty-grey in colour when mature.

Ganoderma, (Karst.) Pat.

Fruit-body bracket-like, corky, covered with a resinous, laccate crust; tubes often stratose; spores truncate, coloured.

Ganoderma lucidum, (Leyss.) Karst. (= *Fomes lucidus* (Leyss.) Fr.)

Fruit-body 5–28 cm. across, chestnut-brown when mature, polished, shining; stem 5–18 × 1–5 cm., lateral, of the same colour as the

pileus; tubes white, then cinnamon; spores elliptical, brown, $10-12 \times 6-8 \mu$.

This fungus appears to be merely saprophytic in temperate regions, but in the tropics it may cause a root disease of the tea bush, bamboo, coconut palm, and other trees. Roots attacked by this fungus are usually covered with tough, thick white strands. In the final stage of attack of the coconut palm the terminal bud decays.

Ganoderma applanatum, (Pers.) Pat. (= *Polyporus (Fomes) applanatus*, Pers.)

Fruit-body 10-40 cm. across, cinnamon with a white margin, bracket-like or imbricate, covered with a laccate crust; tubes ferruginous, 1-4 cm. long, stratose; orifice of pores white, becoming fuscous when bruised, small; flesh cinnamon, firm, thick; spores ferruginous, $9-13 \times 6-8 \mu$.

G. applanatum is one of the commonest bracket fungi on broad-leaved trees such as beech, oak, and maple. It occasionally occurs on conifers. It is a wound parasite, but it attacks only relatively old trees, in which it causes decay of the heart wood. White [7] states that heart wood invaded by this fungus exhibits a mottled appearance and that in living wood the area of attack is delimited by a dark band containing tyloses and copious deposits of gum. Spore discharge from the fructification takes place over a period sometimes as long as six months. Where this fungus is liable to be destructive, losses of timber can be greatly reduced by felling the trees before they are extensively attacked.

In the tropics this species occasionally causes a root disease of tea and coffee.

Ganoderma pseudoferreum, (Wakefield) Overeem and Steinmann

This fungus affects the roots of rubber trees in Malaya and Java, often inducing a wet rot. Diseased roots are sometimes covered with a dark-red skin consisting of mycelium intermingled with host tissues. In advanced stages of decay the wood may be quite spongy. Infection occurs by contact of the rubber roots with diseased roots of jungle stumps.

The fructification may be a small bracket, or it may be hoof-shaped or flattened; the upper surface is brown with a white margin, and the under surface is whitish, becoming yellow when bruised.

Fomes, Fr.

Fruit-body hard, woody, bracket-like or resupinate, sessile, perennial; tubes often stratose; spores white or coloured.

Fomes annosus, Fr.

Fruit-body 7–45 cm. across, deep brown, becoming blackish, imbricate, sometimes resupinate, margin at first white; tubes yellowish, 4–8 mm. long, stratose; orifice of pores whitish, rounded or polygonal; spores sub-globose, white, $4–5 \times 4\,\mu$.

F. annosus causes a serious root disease of conifers and is one of the most important causes of heart-rot in them. It rarely attacks diocotyledonous trees. The fructifications arise around the collar of the tree and upon exposed roots. This fungus is also saprophytic on tree stumps.

The exact mode of infection by this fungus is unknown, but probably small dead roots are first invaded and then the mycelium grows into the larger roots and thence into the collar of the tree, where the mycelium is usually aggregated as a thin white sheet between the bark and the wood. The mycelium has a limited power of growing through the soil by means of fine strands. With young trees the whole of the root system may be killed rapidly by the fungus, when the tree suddenly dies, the only premonitory symptom being a yellowing of the leaves. Older trees are killed outright more rarely, but the fungus on entering the bole of the tree causes a heart-rot which may reduce greatly the amount of marketable timber. In the Scots pine the heart-rot does not generally extend far up the trunk, because of its resinous nature, but in other conifers much of the timber may be rendered worthless. Hiley [8] has studied the effect of the mycelium on the wood of the larch; in the early stages of attack the wood assumes a red-brown colour owing to the formation of gummy substances; black patches appear later in the wood on account of the presence of dark-coloured hyphae, in association with which delignifica-

tion proceeds rapidly, leading to the formation of white patches and eventually holes. The wood generally becomes spongy in character.

In old stands of conifers the trees should be examined with a Pressler borer every few years so that those found to be affected by incipient heart-rot can be felled before much timber is rendered worthless.

According to Anderson [9] heart-rot due to *F. annosus* is most prevalent on acid soils, as the roots in these soils are particularly liable to attack. Coniferous plantations on old agricultural land are also very susceptible to heart-rot, possibly because the drainage of the sub-soil is often deficient, leading to asphyxiation of many of the deeper roots. On markedly acid soils hardwoods rather than conifers should be grown, although in soils too poor for hardwoods the Scots pine may be planted.

Fomes lignosus, Klotzsch

Fruit-body large, red-brown with concentric dark-brown lines, smooth, bracket-like and imbricate ; orifices of pores orange, becoming red-brown ; pores minute.

This is one of the most destructive root parasites of rubber trees, especially on land formerly under heavy jungle. It occasionally attacks tea-bushes and other plants also. The fungus first begins to grow saprophytically on decaying jungle stumps, whence it passes by means of thick, whitish rhizomorphic strands through the soil to living roots in the vicinity. These are invaded and killed, the mycelium travelling back to the collar of the tree, which soon dies. The fructifications usually develop on the stems at about soil level. The rhizomorphs are conspicuous on the dead roots. In rubber plantations the disease is most prevalent in young trees; older trees may also be attacked, but these succumb more slowly. Where this disease breaks out in a young plantation the affected areas should be isolated by means of trenches. In consequence of attack by this and other root parasites, it is now customary to remove all jungle stumps and dead timber as soon as possible after the plantation has been established. This is an expensive undertaking, but is economical in the long run.

Fomes lamaoensis, Murrill   Brown Root Disease of Rubber, Coffee, &c.

Fruit-body large, purple-brown, rather thin, concentrically grooved, bracket-like; orifices of pores dark brown; pores minute.

Fomes lamaoensis attacks the roots of many tropical plants, including rubber, coffee, cocoa, and tea.   The characteristic sign of this disease is the incrustation of affected roots with a brownish mycelium in which particles of soil and small stones are embedded.   The fungus destroys the cortex and attacks the wood, producing brownish bands and lines therein.   The mycelium spreads back to the collar of the tree or bush, where a rot may be induced by it.   The disease progresses somewhat slowly, and the first effect on the shoot system is a thinning of the foliage.   The fungus first develops usually on a dead stump, and, in general, the mycelium infects the roots of cultivated plants only where these come into contact with affected roots of a stump.

The fructifications of the fungus are bracket-like and brown on both surfaces, but the fruit-bodies are rarely seen.

Affected trees should be removed, and if stumps remain in the plantation they should be eradicated.

Fomes igniarius, (Linn.) Fr.

Fruit-body 10–30 cm. across, ferruginous, at length blackish, hoof-shaped; tubes stratose; orifices of pores brown, minute, round; flesh dark brown, very hard; spores globose, white, 5–7 μ.

This is one of the commonest bracket fungi seen on broad-leaved trees, especially oak, beech, ash, poplar, and willow. Infection occurs through wounds.   At first the wood becomes dark-brown in colour owing to the formation of gum, but, after delignification, this is followed by a yellowish-white discoloration, as described by Hubert[4].   *F. igniarius* may be one of the causes of vine 'apoplexy' on the Continent (see p. 267).

Fomes pomaceus, (Pers.) Lloyd

The fructifications are similar to those of *F. igniarius*, but are much smaller, being usually about 5 cm. across.

F. pomaceus occurs chiefly on plum trees, but is occasionally found on cherries. It is usually a slow-growing wound parasite, causing similar changes in the wood to those induced by *F. igniarius.* One side of a plum tree may be covered
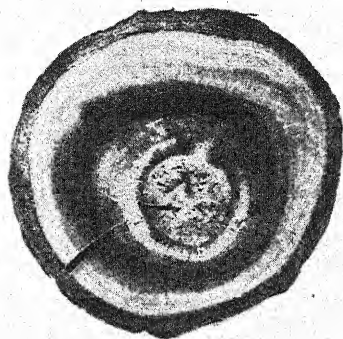
FIG. 53. Branch of a ' Victoria' plum tree affected by *Fomes pomaceus*; the wood recently invaded by the mycelium is discoloured brown, that attacked for a longer time (in centre) is whitish and delignified. Natural size.

with the fructifications, while the other remains healthy. Varieties of plums show marked differences in susceptibility, ' Pershore' being one of the most liable to attack; ' Victoria' is usually very resistant to *F. pomaceus*, but where branches of this variety are broken by heavy crops this fungus may invade the wounds and cause a rapid die-back.

Fomes fomentarius, (Linn.) Fr.

Fruit-body 10–60 cm. across, greyish, hoof-shaped; tubes brown, stratose; orifices of pores finally brown, minute, round; spores elliptic-oblong, white, $16–18 \times 5 \mu$.

The fleshy part of the fruit-bodies of this fungus was formerly used as tinder. It occurs commonly on the beech, oak, poplar, and birch, in which it causes a wood rot.

Fomes Ribis, (Schum.) Fr.

The dark-brown fruit-bodies of this fungus, which are yellow at the margin, are found on the stems of old currant and gooseberry bushes.

Polyporus, (Micheli) Fr.

Fruit-body fleshy or coriaceous, bracket-like or rarely resupinate, chiefly annual ; tubes not stratose ; spores white or coloured.

Polyporus adustus, (Willd.) Fr.

Fruit-body 3–7 cm. across, brownish-grey, becoming black at the margin, obsoletely zoned, bracket-like and often imbricate ; orifice of pores at first white and then blackish, minute ; flesh 3–5 mm. thick ; spores elliptical, white, 4–5×2–$3\,\mu$.

Although this fungus is usually saprophytic, occurring commonly on tree stumps, Prior [10] has recorded it as a parasite on branches of beech trees, and Brooks [11] as a wound parasite of apple trees. In apple trees the fungus enters chiefly through large wounds made in thinning out branches, from which it spreads rapidly downwards. The bark as well as the wood is killed by the growth of the fungus, and one of the marked symptoms of attack is the cracking of the bark. Large wounds in such trees should be covered with soft grafting wax or with a thick paint (see p. 272).

Polyporus hispidus, (Bull.) Fr.

Fruit-body 10–30 cm. across, ferruginous and finally blackish, very hispid, imbricate ; orifices of pores yellowish, becoming brown, small, becoming torn ; flesh brown, 2·5–10 cm. thick ; spores subglobose, brown, 9–10×7–$8\,\mu$.

This occurs commonly on the trunks of ash, elm, and apple trees. Large quantities of water are often exuded from the under-surface during active growth of the fructifications.

Polyporus betulinus, (Bull.) Fr.

Fruit-body 7–30 cm. across, silvery-grey, becoming brownish, smooth, bracket-like ; orifices of pores white, becoming darker, minute, round ; flesh white, soft, then corky. Spores oblong, white, 5–$7 \times 2\,\mu$.

Birches are often killed by this fungus, which causes a white rot of the wood. Trees attacked by it frequently break off at a height of about 10 feet.
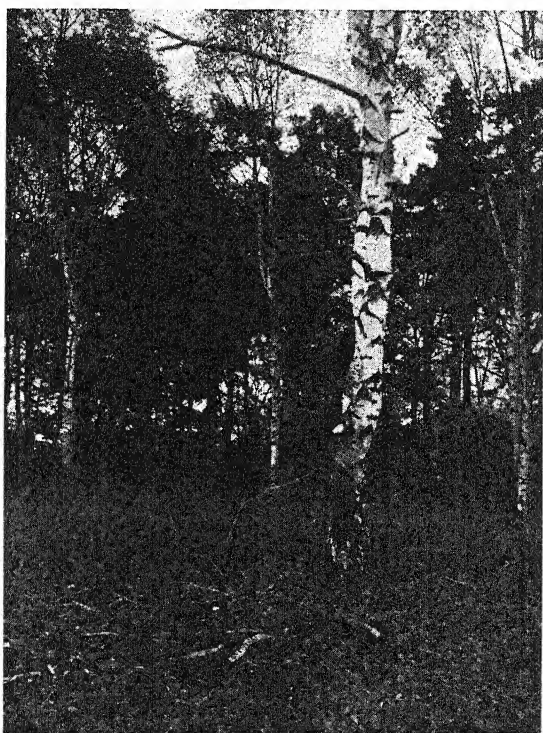
FIG. 54. Birch tree killed by *Polyporus betulinus*, showing fructifications.
(*A. Smith.*)

Polyporus Schweinitzii, Fr.

Fruit-body 10–40 cm. across, brown, tomentose, circular in outline or bracket-like, usually with a short stem ; orifices of pores greenish yellow, broad, often irregular ; flesh soft, becoming brown ; spores elliptical, white, $7–8 \times 4 \mu$.

A heart-rot of conifers is caused by this fungus, which often enters the bole by way of the roots. It is fairly common on Scots pine and larch in Great Britain, and attacks conifers in N. America, where it sometimes enters the trees through

fire-scars. The exact mode of infection of the roots is obscure. The fructifications appear around the bole of the tree or arise from the larger roots. Wood rotted by this fungus is dry and friable, and tends to break up into more or less cubical blocks, the interstices being often filled with white mycelium. The individual tracheids, which are partly delignified, show oblique cracks owing to contraction. Trees bearing fructifications should be felled, as in most of them a heart-rot of the bole will have already begun.

Polyporus sulphureus, (Bull.) Fr.

Fruit-body 10–40 cm. across, orange, imbricate, usually sessile; orifices of pores sulphur yellow, minute, round; flesh cheesy, light yellow, becoming white; spores elliptical, white, $7-8 \times 5 \mu$.

This is one of the few polyporoid fungi which attack both broad-leaved trees and conifers, although in Britain it is most common on the former, especially on willow, poplar, and oak. Infection occurs through wounds above ground, and results in a heart-rot of the wood, which becomes reddish brown, the annual rings exhibiting a tendency to separate one from the other. Fructifications arise during the summer in dense, imbricated masses from wounds on the trunk.

Polyporus squamosus, (Huds.) Fr.

Fruit-body 10–60 cm. across, ochraceous, covered with darker brown scales, bracket-like and sometimes imbricate; stem short, excentric, blackish at the base; orifices of pores pallid, large, angular; flesh white; spores oblong, $10-12 \times 4-5 \mu$.

This species is very common in England on most kinds of dicotyledonous trees, particularly elm. It is a wound parasite which causes a white heart-rot of the wood, the sap wood being affected last. The wood is ultimately rendered quite soft. Trees affected by P. squamosus are often blown over during gales, owing to loss of rigidity caused by delignification. Elm trees bordering thoroughfares should be tested from time to time, and if the heart wood is found to be decayed they should be felled in order to avoid danger to traffic. Buller [12] has described the effect of the fungus on sycamore wood. The fibres between the vessels are the first to be delignified, and

subsequently all other elements of the wood are more or less delignified, the wood generally becoming much lighter in colour and tending to split into irregular cubes. The hyphae pass readily from cell to cell, often boring passages for themselves.
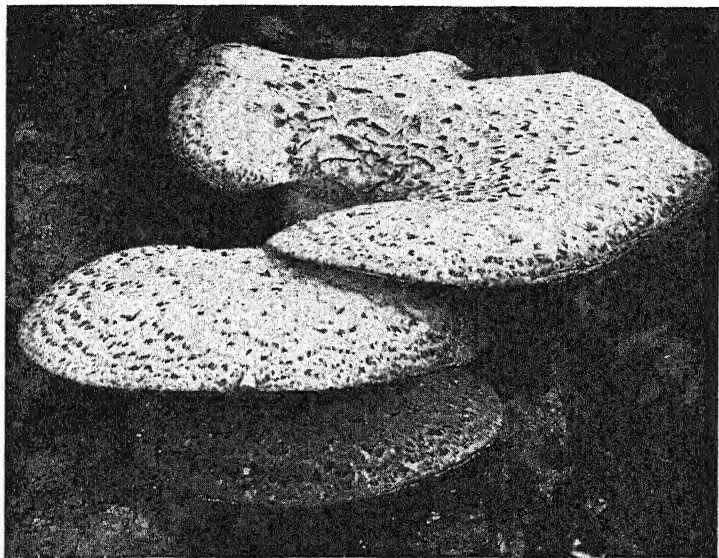
FIG. 55. Fructifications of *Polyporus squamosus*, $\frac{1}{5}$ natural size. (*Somerville Hastings.*)

The fungus usually takes a long time to kill a tree, but before death ensues fructifications arise on the trunk during the summer. Buller[13] and Brooks[14] have pointed out that a spore cloud, which can be seen clearly in a dim light, is continuously emitted from the fructifications for more than a week.

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FUNGUS DISEASES (*continued*): AGARICALES

AGARICALES

BASIDIA non-septate, spores (usually four) formed at the apex, at the extremity of slender sterigmata. Hymenium at first covered by a volva or a ring, becoming fully exposed at maturity and spread usually over the surface of gills. The fruit-bodies are large and often mushroom-like. Clamp connexions often occur in the vegetative mycelium. The only family of pathological importance is the Agaricaceae, in which the hymenium is inseparable from the pileus and is spread over the surface of gills.

Lentinus, Fr.

Pileus coriaceous, stalked or sessile ; stem excentric ; gills tough, adnate or decurrent, often toothed at the edge ; spores white.

Lentinus lepideus, Fr.

This fungus sometimes causes a rot of wood-paving blocks, timber used in bridges, and railway sleepers, although it does not usually attack these, if creosoted, until after their ' mechanical life ' is exhausted. The destruction of wood-paving has been described by Buller [1]. The fruit-bodies are stalked and rigid, and the pileus is covered with dark scales.

Marasmius, Fr.

Pileus membranaceous or coriaceous, regular or resupinate, stem central or absent ; gills adnate, adnexed, decurrent or free, rather tough ; spores white.

Marasmius oreades, (Bolt.) Fr.

Fairy-rings in poor pastures are often caused by this fungus. Three zones are usually seen in a fairy-ring in summer : an inner zone in which the grass is stimulated, a middle one in which the grass is dead or very weak, and an outer zone in which the grass is also stimulated. The outer zone, however, is often alone conspicuous. Fructifications appear in the

central zone and often also in the outer zone in summer and autumn. The fruit-bodies are buff in colour, and are about 3 cm. across. According to Bayliss-Elliott [2] the mycelium is both saprophytic in the soil and parasitic on the grass roots; it is responsible for the death or weakening of the grass in the middle zone. The mycelium dies out in the central region, and the grass is stimulated by the additional supplies of nitrogen available on the decomposition of the mycelium. The outermost zone of the ring is also stimulated by additional supplies of nitrogen made available through the growth of the fungus in the soil. Several other fungi form fairy-rings in pastures. In N. America, according to Shanz and Piemeisel, [3] the death of the grass in the middle zone of fairy-rings caused by *Agaricus tabularis* is due to desiccation induced by inability of water to percolate through the soil owing to the abundant mycelium present.

Marasmius Sacchari, Wakker

Sugar-cane often suffers from root disease, one of the causes of which is *M. Sacchari.* The stools are at first stunted, and the roots and basal joints become rotten, the fructifications appearing as small toadstools around the latter. According to Nowell [4] the disease is intimately associated with environmental conditions, predisposing factors being drought, poor soil, imperfect aeration of the soil, and insect attack. It is difficult to assess the actual loss caused by this fungus, but it is undoubtedly weakly parasitic throughout the cane-growing regions of the West Indies, and it may sometimes be much more virulent. Where the crop is grown under good conditions of cultivation, with an extended rotation, losses from this disease are reduced to a minimum.

Marasmius equicrinis, Mull.

'Horse-hair blight' is the popular name given to the black festoons of mycelium which hang from the branches of tea bushes and other plants in wet regions of the tropics. The mycelium is attached to the shoot at intervals by small disks, but is not actually parasitic. The stalks of the brownish fructifications are black.

Marasmius pulcher, (B. and Br.) Petch

This white 'Thread Blight' fungus is entirely epiphytic, and occurs commonly on tea, rubber, and other tropical plants. The strands of mycelium spread indefinitely over the stems and leaves, sometimes matting the latter together. The fructifications, which are found only on fallen leaves, are very minute.

Another 'Thread Blight' fungus of tea in Assam and Ceylon is undoubtedly parasitic, but its fructifications have not yet been found. Here the threads expand into a delicate film as the under surface of the leaves is reached, and the tissues soon die. Considerable damage may be caused by this fungus. All twigs bearing the white threads should be cut out, and older branches, if affected, should be sprayed with lime-sulphur.

Pleurotus, Fr.

Pileus fleshy; stem excentric or wanting; gills adnate or decurrent; spores white, or rarely pinkish or lilac.

Pleurotus ostreatus, (Jacq.) Fr.   Oyster Shell Fungus.

This is a weak wound-parasite of broad-leaved trees, although no details of its parasitism are available. The fructifications are yellowish-white, 7–13 cm. across, and are often devoid of a stem; the spores are lilac in mass.

Several other species of Pleurotus are occasionally parasitic on trees.

Collybia, Fr.

Pileus fleshy, regular, margin incurved; stem central, cartilaginous; gills adnate, adnexed or free; spores usually white.

Collybia velutipes, (Curt.) Fr.

This common toadstool is occasionally weakly parasitic on trees which have been cut back, e. g. laburnums, and on old red currant bushes. The fructifications, which often arise in groups, are tawny with a darker coloured stem, which is velvety towards the base. Unlike most fungi of this class, the fruit-bodies can withstand several degrees of frost, so that they often develop in Britain throughout the winter.

Clitocybe, Fr.

Pileus fleshy, margin incurved; stem central, externally fibrous; gills decurrent; spores usually white.

Clitocybe parasitica, Wilcox

In Missouri and neighbouring parts of the United States this fungus is a destructive root parasite of apple and other trees, according to Wilcox[5]. The fungus spreads by means of rhizomorphs, and the fruit-bodies arise in dense clusters around the base of the tree. In other districts the fungus appears to be only saprophytic.

Pholiota, Fr.

Fruit-body fleshy; stem central with a ring; gills adnate; spores ochraceous or ferruginous.

Pholiota squarrosa, (Müll.) Fr., is sometimes weakly parasitic on the trunks of trees, especially ash and lime. The fructifications are densely caespitose, ochraceous, and covered with persistent scales on the upper surface.

Armillaria, Fr.

Pileus fleshy, regular; stem central, fleshy, provided with a ring, gills sinuato-adnexed or slightly decurrent; spores white.

Armillaria mucida, (Schrad.) Fr.

This species is weakly parasitic on beech trees; it occasionally occurs also on oaks and birches. The fructifications are glistening white, diaphanous, and slimy to the touch.

Armillaria mellea, (Vahl) Fr.   Honey Fungus.

Pileus 5–10 cm. across, tawny; stem tawny with a white ring that becomes discoloured with age; gills whitish, then rufescent, adnate or slightly decurrent; spores $8-9 \times 5-6\,\mu$. The fructifications are often caespitose and are extremely variable.

A. mellea is perhaps the most dangerous subterranean parasite of trees, bushes, and certain herbaceous plants. One of its many forms is probably the cause of a serious root disease of cocoa trees in the Gold Coast. Conifers are particularly susceptible to attack, but the larch does not usually succumb under the age of fifteen years. Broad-leaved trees are often

killed by this fungus, and such plants as privet, gooseberry, strawberry, and even the potato and rhubarb, may be affected.

It is likely that *A. mellea* nearly always begins life as a saprophyte upon decaying tree stumps. From an affected stump the fungus grows out in the form of rhizomorphic

FIG. 56. Fructifications of *Armillaria mellea* arising from the base of a tree which it has killed. ⅛ natural size. (*Somerville Hastings.*)

strands (*Rhizomorpha subterranea*), which spread indefinitely a few inches to a foot or more below the surface of the soil. These rhizomorphs look like black boot-laces and show considerable internal differentiation. Rhizomorphs appear not to exist in the form of the fungus that occurs in the Gold Coast. When the rhizomorphs come into contact with the roots of a living tree, they penetrate the tissues and give rise to vegetative mycelium of the ordinary kind, which spreads chiefly along the line of the cambium back to the collar of the tree, whence other roots are affected. It is probable that only injured or dead roots can be invaded by the rhizomorphs, but

this is uncertain. When the collar has been girdled by the mycelium or if practically the whole root system has been invaded, the tree suddenly dies. In the bark and along the zone of the cambium the mycelium is present in the form of dense white sheets, and as the wood shrinks away from the dead bark a network of rhizomorphs (*Rhizomorpha subcorticalis*) is differentiated. The mycelium also penetrates the wood, causing partial delignification. A fairly common feature of the invasion of the wood is the formation in it of narrow black lines consisting of thick-walled cells of the fungus. *A. mellea* causes a serious butt-end rot of *Populus tremelloides* in N. America and of *P. tremula* in Sweden. The fructifications arise from the base of the tree and are attached to rhizomorphs; the fruit-bodies may also arise from rhizomorphs in the soil.

Whenever trees and bushes are planted in land containing stumps there is risk of attack by *A. mellea*. If economically possible, all stumps should be removed, but in most forestry operations this is not practicable. Alternatively, the stumps left in newly planted land should be watched, and when these are seen to give rise to fruit-bodies of the fungus in the autumn they should be removed, as it is from these stumps that the infecting rhizomorphs derive their food. Similarly when trees or bushes are killed by *A. mellea* they should be removed in order to prevent further infection.

The fungus is usually most destructive in badly drained soil, but there is need for further investigation into its ecology. In mixed plantations this parasite is often specific in its attack. The author once saw a mixed plantation of ash and cricket-bat willow in which the latter alone was affected.

An extraordinary association of *A. mellea* with the tubers of the orchid *Gastrodia elata* has been described by Kusano[c]. Here the tubers may be destroyed by the invading rhizomorphs, but the mycelium, after penetrating the tubers, is sometimes confined to a certain zone through digestion of the fungus by the host cells, so that a kind of symbiosis is established. It is only such partly invaded tubers that flower.

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FUNGUS DISEASES (*continued*): SPHAEROPSIDALES

SPHAEROPSIDALES

THIS group comprises those fungi which possess pycnidia, but in which ascus fructifications have not yet been found. In recent years many fungi, formerly placed in the Sphaeropsidales, have been found to be Ascomycetes on fuller investigation. Some of those described here may be similarly classified in the future.

Certain genera in this group are ill-defined. For instance, there is no valid distinction between Phoma and Phyllosticta, and no sharp line can be drawn between Phoma and Diplodina.

Phoma, (Fr.) Desm.

Pycnidia at first covered by the epidermis of the host, then erumpent, membranous, globose or lenticular, opening by a pore ; conidiophores short or almost absent ; spores unicellular, hyaline, usually exuding from the pycnidium in a worm-like manner.

The only distinction between Phoma and Phyllosticta is that in the former pycnidia are formed on stems and in the latter on leaves. Occasionally, too, some of the spores are uniseptate.

Phoma Betae, (Oud.) Frank   Heart Rot of Sugar-Beet and Mangolds.

This fungus causes a serious disease of sugar-beet and mangolds on the Continent, and it also occurs in England. The disease was first described by Frank [1], who pointed out that the fungus passed from the blackened heart leaves into the root, where a rot was set up. Plants grown for seed are also affected. The same fungus is partly responsible for the rotting of mangolds in storage, although many other factors play a part in causing losses of this kind. A damping off of seedling mangolds and sugar-beet in England, associated with a blackening of the stem ('Black-Leg'), has recently been recorded in the Plant Disease Survey of the Ministry of Agriculture, and one of the causes of this disease is *Phoma Betae,* which is often carried over on the 'seed'.

The pycnidia are formed on all the affected parts of the plant, but are most abundant on the leaves, leaf-stalks, and 'seed'. They are about $200\,\mu$ in diameter and the spores measure $4 \times 3\,\mu$.

There is a difference of opinion as to the part actually played by the fungus in causing 'Heart Rot'; some authorities consider that only beets and mangolds grown under certain conditions are susceptible to attack. Gäumann[2] in a recent monograph states that in Switzerland *P. Betae* is only capable of attacking plants grown in markedly alkaline soils.

In Holland[3] the 'seed' is now disinfected on a large scale by immersing it for 3–5 hours in a 0·25 per cent. solution of copper sulphate in water at 42° C. and subsequently drying it. In Germany it is recommended to steep the 'seed' in 0·5 per cent. carbolic acid.

Phoma Lingam, (Tode) Desm. (= *P. Napobrassicae*, Rostr. and *P. oleracea*, Sacc.)   Dry Rot of Swedes and Turnips; Cabbage Black-Leg.

Pycnidia globose, lenticular or flask-shaped, brownish-black, 130–340 μ; spores elliptical, sometimes slightly curved, ends bluntly rounded, unicellular, hyaline, 3·5–6 × 0·8–2 μ.

Dry-rot of swedes and turnips is the most serious disease of these crops in New Zealand, where it has been studied by Cunningham[4]. It occurs also in Britain on swedes grown for food and for seed, in other parts of Europe, and in North America.   The same fungus causes a stem rot of Brassicae in Britain and the 'Black-Leg' disease of cabbage in the United States, described by Henderson[5].

The disease is seed-borne. Upon germination the fungus passes from the testa to the cotyledons, forming brown lesions in which pycnidia occur. From these, spores infect the leaves and stems, sometimes producing a 'damping-off' effect. With swedes and turnips spores are washed down to the 'bulbs', the entire surface of which may become covered with brownish, depressed lesions. The fungus also spreads to the leaves of the flowering spikes and the fruits, whence the seed is infected.

The control of this disease lies in the production of healthy

seed, as it is difficult to disinfect the seed satisfactorily. If mother plants could be grown from clean seed, the resulting crop of seed would probably be free from disease. Walker [6] advocates the treatment of cabbage seed with hot water at 50° C. (122° F.) for 30 minutes, although this reduces germination somewhat. Cunningham [4] found this method unsatisfactory with swede seed, and he advises immersion in 0·25 per cent. semesan at 115° F. for 1 hour. The reason why the disease should be so much more serious in New Zealand than in Britain is not fully understood : it may be accounted for by differences in climate, soil, or manurial treatment. The seed used in New Zealand comes from England.

Phoma apiicola, Klebahn

This was first described by Klebahn [7] as the cause of a disease of celeriac, but it also causes a rot of the roots of celery seedlings, a rot of the lower parts of older celery plants, and occasionally a spotting of the leaves. Pycnidia occur chiefly on the underground parts. The disease has recently been recorded in England on celery, but it is of little consequence here.

Phoma destructiva, (Plowr.) C. O. Jamieson

This species causes a rot of tomato fruits as they approach maturity, and, according to Jamieson [8], it also affects the stems and leaves in the United States. Brooks and Searle [9] point out that it is doubtful whether a sharp line can be drawn between this species and *Diplodina Lycopersici,* Hollós, the pycnidial stage of *Didymella Lycopersici,* Klebahn.

Phoma Lavandulae, Gab.

The cultivation of lavender on a large scale in this country for making lavender oil is threatened by a disease, known as ' Shab ', which, according to Brierley [10], appears to be due to this fungus. The first symptom of attack is the wilting of the young shoots of one part of the bush, which is followed by the death of this part and later of the whole bush. The disease spreads rapidly. The pycnidia are formed chiefly on the young

twigs and occasionally on the leaves; the spores measure
$4 \times 2\ \mu$.   The disease is at present under further investigation
at Cambridge.

FIG. 57. Lavender bush affected by 'shab'.   (*C. R. Metcalfe.*)

Phoma Pomi, Passerini

This fungus causes a spotting of apple fruits in the eastern
United States, New Zealand, and Europe.   As the fruit colours,
the spots, which are always small, become conspicuously
surrounded by a reddish margin, but during storage the spots
become brown and somewhat sunken.   The first kind of
fructification is of the Cylindrosporium type (*C. Pomi*, Brooks),
but pycnidia with unicellular, hyaline spores are formed later.
The disease, which is usually of little importance, can be
controlled by spraying with lime-sulphur of spec. grav. 1·003
just before the fruit matures.

Phoma tuberosa, Melhus, Rosenbaum, and Schultz, causes a dry rot
of potato tubers in the United States, where Melhus, Rosenbaum,

and Schultz [10a] state that it is found sometimes in association with *Spongospora subterranea.* It occurs occasionally in the British Isles.

Phyllosticta, Persoon

The characters are the same as those of Phoma, but the pycnidia are formed on leaves instead of stems.

Phyllosticta prunicola, Sacc.

This is one of the fungi which produce a shot-hole effect in the leaves of stone-fruit trees. Small portions of the leaf tissue are turned brown by the mycelium, which spreads only to a limited extent. The diseased tissue is sharply delimited so that it falls away leaving a hole. The pycnidia are usually formed only on the portions which fall to the ground.

Phyllosticta solitaria, E. and E., produces a blotch of apple fruits in the southern part of the United States, and has been investigated by Scott and Rorer [11].

Phyllosticta Antirrhini, Syd.

Buddin and Wakefield [12] describe this species as causing considerable damage to the stems and leaves of Antirrhinums. When the stem is attacked towards the base the plant frequently dies. The leaf spots are pale in colour and later dry out. The capsules also may be affected, and portions of these bearing pycnidia may be included with the seed. The fungus may also pass the winter as pycnidia on dead stems and leaves. The disease is most prevalent under greenhouse conditions.

Phomopsis, Sacc.

Characters mostly as in Phoma, but the pycnidia often contain curved, filiform, hyaline ('b') spores (?) as well as Phoma-like ('a') spores; the pycnidial wall is thick and stromatic, and the conidiophores are more permanent than in Phoma.

Phomopsis Pseudotsugae, Malcolm Wilson

Pycnidia globose-depressed, often in groups of two or three, incompletely or completely chambered, black, 0·1–0·5 mm. across;

spores of one kind only ('a' spores), elliptic-fusoid, obtuse at the
ends, $5\cdot5-8\cdot5 \times 2\cdot5-4\,\mu$.

According to Wilson [13], this fungus may prove to be the pycnidial
stage of *Diaporthe pithya*, Sacc.*

This fungus causes a serious disease of the Douglas fir in
Britain, and it more rarely attacks the Japanese larch and
certain other conifers. Our knowledge of the disease is chiefly
due to Wilson [13].

One effect of the fungus is to kill back for some distance the
leader shoots or the young parts of lateral shoots. In this case
infection may be brought about in uninjured tissues before
cork has been formed, or it may occur through wounds in
older tissues. Shoots so affected may be killed back for
a distance of nine inches, the dead bark being separated from
the living tissues by a layer of cork. Pycnidia are formed in
large numbers on these dead shoots.

The fungus may also attack the main stem lower down,
frequently near soil level. This it does either through
abrasions or by first infecting a lateral shoot, from which the
mycelium enters the main stem. In young trees the mycelium
frequently grows throughout the bark at this level, and causes
the death of the upper part of the tree. In this kind of attack
the stem just above the infected region is considerably swollen.
In older trees infection of the lower part of the main stem
more frequently results in the formation of a canker, which
may gradually bring about the death of the tree or which
may be ultimately occluded by the formation of callus.

The Douglas fir is most liable to attack between the ages of
4 and 15 years. The disease occurs both in the nursery and
after planting out. Diseased plants in the nursery should be
burnt, and young trees which are seriously injured in the
plantations should also be destroyed. Care should be taken in
pruning the branches of older trees to avoid wounding the
main stem, as such wounds often result in the formation of
cankers. Suppressed trees are often liable to attack by this
fungus; they should be removed if affected by it.

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* This ascigerous fungus is often found as a saprophyte on shaded-out
branches of the Douglas fir.

Phomopsis cinerescens, (Sacc.) Trav.   Fig Canker.

Pycnidia lenticular, 250–300 μ in diameter; spores ellipsoidal to fusiform, often with one end more rounded and broader than the other, hyaline, 6·5–13 × 2·4–3·6 μ.

This is the cause of a serious canker of the branches of fig trees on the Continent and in England, as described by Salmon and Wormald[14]. The fungus is a wound parasite, and the cankers often extend to such an extent that the branches above them are killed.

Phomopsis Citri, Fawcett

Several species of Phomopsis cause stem-end rots of citrus fruits, of which *P. Citri* is the commonest on oranges and grape-fruit. The same fungus kills the twigs and causes a black spotting of the fruit ('melanose'). *P. Citri* may attack fruit on the tree, but is more often evident as the cause of a rot in storage. Infection usually occurs at the 'button' or stem end, where decay begins as a leathery zone, pale yellow to brownish in colour. Within the fruit the rot extends most rapidly in the middle. Pycnidia rarely occur on the fruit, but are commonly found on twigs. The 'a' spores measure 5–9 × 2·5–4 μ, and the 'b' spores 20–30 × 0·75–1·5 μ. Wolf[15] has found the perfect stage, *Diaporthe Citri* (Fawcett) n.sp., on dead twigs of lime and orange.

Fawcett and Lee[16] recommend removal of dead twigs and spraying of the young fruits with Bordeaux mixture to which 1 per cent. paraffin emulsion * has been added. The fruit should be stored at a temperature of 40–45° F.

Macrophomina, Petrak

Pycnidia as in Phoma, but the spores are long and narrow.

Macrophomina Phaseoli, (Maubl.) Ashby (= *Rhizoctonia bataticola*, (Taub.) Butl.)

The synonymy of this fungus has been dealt with by Ashby[17]. It is one of the commonest root parasites in warmer lands, and is omnivorous, attacking jute, groundnuts, cotton, runner beans, sesame, sweet potatoes, and perhaps also coffee, cocoa, tea, and rubber. The mycelium spreads through the soil. The sclerotia, which are smooth, hard and black, and 50–1,000 μ in diameter, occur chiefly on roots that have been attacked. The pycnidia, which are not invariably present, are formed in the stems; the spores are very variable in size, ranging from 16–30 × 5–10 μ. The fungus causes a seedling blight, and stem and root rot of herbaceous plants, and perhaps also a root decay of woody plants, as suggested by Small[18].

* See page 363.

Polyopeus, Horne

The genus is distinguished from Phoma because each pycnidium is provided with one or several necks, at any rate when the fungus is grown on culture media.

Polyopeus purpureus, Horne

In some seasons this fungus causes much 'spotting' and rotting of apples in England during the early period of storage. The rot usually begins at a lenticel, as described by Kidd and Beaumont [19]; the diseased tissues, which are brown with a purplish tinge, are often smooth and circular in outline. The fungus rarely fruits on the apple, but pycnidia are formed abundantly in culture. Several other species of Polyopeus occasionally cause 'spotting' and rotting of stored apples.

Cytospora, Ehrenb.

Pycnidia formed in or on a sharply defined stroma; spores sausage-shaped, small, unicellular, hyaline. Some species belong to *Valsa* spp. See page 201 *et seq.*

Coniothyrium, Corda

Pycnidia subcortical or superficial, globose or depressed, ostiole papillate, black; spores small, globose or ellipsoid, brown; conidiophores short or absent.

Coniothyrium rosarum, Cooke and Harkness

Grafted roses are often attacked by this fungus, which gains entrance at the junction of stock and scion. The disease has been investigated by Vogel [20] in the United States. The lesions first appear as yellow, water-soaked areas, which become brown. The plants may rapidly wilt and die, but sometimes a large canker is formed, in which case a dwarfed, one-sided plant develops. The fungus fruits on the dead tissues. The Manetti rose stock is very resistant to the disease, but susceptible varieties grafted on it are often attacked. Shoots for scions should not be taken from roses on which the disease exists. *C. Fuckelii* (see p. 190) also causes a serious canker of rose stems.

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Ascochyta, Libert

Pycnidia occurring chiefly on spots on leaves and stems, globose-lenticular, with an ostiole ; spores ovate, uniseptate, hyaline.

Ascochyta Violae, Sacc. and Speg., forms large, brownish or yellowish patches on the leaves of cultivated violets. The spores measure $10–11 \times 3–4 \mu$.

Ascochyta Pisi, Lib., emend. Linford and Sprague   Pea Spot.

Pycnidia on definite spots, light to dark brown, $75–225 \mu$; spores oblong with rounded ends, uniseptate, somewhat constricted, hyaline, $10–14 \times 3–5 \mu$.

This fungus attacks almost any part of the shoot system of peas, and it has been recorded occasionally on other leguminous plants. The fungus is commonly carried over in the seed coats, and spores liberated during germination may infect the seedlings. Older stems, leaves, and pods are often attacked, the fungus forming grey or brown, sunken spots, which bear pycnidia. The fungus grows through the pod into the seeds.

Epidemics of this disease occur only in wet seasons. There is considerable difficulty at present in obtaining seed which is entirely free from this fungus. As far as possible peas for seed should be harvested only from sound pods.

Vaughan [21] considered that the perfect stage of this fungus was *Mycosphaerella pinodes*, (Berk. and Blox.) Niessl, but Linford and Sprague [22] state that *M. pinodes*, which affects peas similarly and causes a foot-rot in addition, is a different fungus.

Diplodina, Westendorp

Pycnidia and spores as in Ascochyta, but the pycnidia do not develop on spots.

Diplodina parasitica, (Hartig) Prill., affects the twigs and leaves of the common spruce in nurseries and young plantations, causing defoliation. The spores measure $13–15 \times 3–4 \mu$.

Diplodia, Fries

Pycnidia black with an ostiolate papilla ; spores ellipsoid or ovate, uniseptate, brownish-black at maturity ; conidiophores needle-shaped, simple, hyaline.

Diplodia natalensis, Evans

Pole Evans [23] has described a rot of citrus fruits caused by this fungus in South Africa. A similar disease occurs in other citrus-growing countries, but more than one species of Diplodia may be involved. The rot chiefly affects the fruit during storage and transportation, but it occasionally attacks the fruit on the tree. The fungus usually enters the fruit at the stalk end and causes a leathery decay, the fruit gradually becoming blackish. If the fruit is kept in a moist atmosphere it becomes covered with a dark mycelium. The fungus also occurs on dead twigs and bark of citrus trees, on which pycnidia are formed in abundance. The spores measure $24 \times 15 \mu$, and the walls are ornamented with striated bands. Stevens [24] indicates that the ascospore stage of the Diplodia causing stem-end rot of citrus fruits in Florida is *Physalospora rhodina*, (B. and C.) Cooke, and that the fungus occurs on several other hosts.

Dead wood likely to harbour the sporing stages of the fungus should be cut out in citrus orchards; the fruit should be kept at a low temperature during storage, the duration of which should be shortened as much as possible.

Diplodia gossypina, Cooke, causes a rot of cotton bolls in Louisiana, the West Indies, and other cotton-growing countries. According to Edgerton [25] it obtains entry through insect punctures or other wounds, and causes a rot of the interior before the outside of the boll is much affected. The fungus is closely related to *D. natalensis*. Stevens [26] states that the perithecial stage is a Physalospora, which he names *P. gossypina*. The pycnospores do not become coloured until ready for discharge ; they measure 17–35×9–23μ.

Diplodia Zeae, (Schw.) Lév.   Dry rot of Maize.

This disease commonly affects maize cobs in the United States and in South Africa, causing shrivelling and discoloration, and a considerable reduction in yield. Even grain which appears sound may be invaded by the mycelium, and such grains either do not germinate or give rise to weakly plants. Infection occurs after flowering, around the sheaths or through the stigmas. The fungus also produces reddish spots on the leaf sheaths, and persists as a saprophyte on the dead remains of the maize plants. Durrell [27] points out that the disease is greatly favoured by heavy rain as the

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crop is approaching maturity. Care should be taken to use only seed grain which is free from this fungus.

The pycnospores are usually slightly curved and measure 24–33 × 5 μ.

Botryodiplodia, Sacc.

Pycnidia often grouped together in an erumpent stroma, otherwise as in Diplodia.

Botryodiplodia Theobromae, Pat.

Pycnidia globose, 250–400 μ in diameter; spores oval, uniseptate and blackish brown at maturity, 25–35 × 14–15 μ; paraphyses linear, 40–80 μ long.

This fungus has received many names and is sometimes referred to the genus Diplodia.

This is one of the commonest saprophytes in the tropics, but it is also a dangerous wound parasite of rubber, cocoa, tea, and other tropical plants, causing a rapid die-back. With rubber trees the fungus enters either through a wound (as when it attacks newly-planted 'stumps') or through tissues already killed by some other fungus. Young twigs killed by *Gloeosporium alborubrum* or *Phyllosticta ramicola*, and older branches attacked by *Corticium salmonicolor*, are often secondarily invaded by *B. Theobromae*. The fungus grows downwards in the wood with great rapidity, killing large branches and sometimes entire trees. The wood becomes blackish owing to the dark mycelium. The pycnidia, formed just under the surface of the bark, may become covered with a black dust on extrusion of the spores.

B. Theobromae may cause a serious root disease of tea. According to Petch [28] the fungus generally develops first on tea prunings buried in the ground, whence it attacks the roots. The young leaves become mottled with yellow and then turn black and fall. The symptoms may extend from one branch to another until the whole bush is dead. Where the disease is likely to occur in tea the prunings should be burnt.

Stagonospora, Sacc.

Pycnidia globose, ostiolate, brownish-black; spores elliptical or elongate, 2 or more septate, hyaline.

Stagonospora Curtisii, (Berk.) Sacc., sometimes causes brown spots on Narcissus leaves, especially near the tips. It has often been recorded on the variety Soleil d'Or in the Scilly Isles. The spores are unicellular or 1–3 septate, and measure $7-26 \times 5-7 \mu$. *S. Narcissi*, Hollós, is probably the same fungus.

Septoria, Fries

Pycnidia usually on leaf spots, globose-lenticular, ostiolate, brownish-black ; spores narrowly elongate to filiform, multiseptate, hyaline ; conidiophores very short.

Septoria Apii, Chester   Celery Leaf Spot.

Pycnidia 180–250 μ in diameter ; spores often slightly curved, usually 3-septate, $50-65 \times 1 \cdot 5 \mu$.

'Leaf Spot' attacks both seedling and adult celery plants, and is sometimes responsible for great losses. Pethybridge [28a] has recorded the fungus on Wild Celery in Ireland. Pycnidia are often found on the celery 'seed', and it is chiefly in this way that the disease is disseminated. The spores from these pycnidia attack the seedlings, and if slightly affected young plants are set out the disease spreads epidemically in wet weather. Small, scattered brown spots appear on the

FIG. 58. Section through pycnidium of *Septoria Apii*. × 215. (*W. J. Dowson.*)

leaves, which may increase until the entire foliage is killed, the pycnidia being formed in the dead tissues. Second year celery plants, kept for seed, are also attacked, and pycnidia may often be seen on the young fruits.

The best means of avoiding the disease is by the use of clean seed. Where this cannot be guaranteed, the seed should be steeped for three hours in weak formalin (1 part commercial formalin to 400 water) ; it is important that the seed should be thoroughly wetted by stirring or shaking in order to get rid of adherent air. If the disease appears in field crops it can be

controlled by spraying several times with Bordeaux mixture. Clean 'seed' may possibly be obtained by spraying the plants in the flowering and 'seeding' stage.

Septoria Paeoniae, West., causes a leaf blotch of paeonies.

Septoria Dianthi, Desm., sometimes attacks the leaves and stems of carnations under glass, producing light brown spots. Where there is good ventilation and careful watering the disease is not likely to be serious.

Septoria Violae, West., causes a leaf spot of violets.

Septoria Lycopersici, Speg., causes a serious leaf blight of tomatoes in the United States and the Argentine, but it has been recorded only occasionally in England. The disease can be checked by spraying with Bordeaux mixture early in the season.

Septoria Antirrhini, Desm., forms pale, round spots, with a purplish margin, on the leaves of Antirrhinums during cool, wet weather.

Septoria chrysanthemella, Cav., causes brown blotches on the leaves of chrysanthemums. The leaves curl and fall prematurely. Other species of Septoria also attack this host.

Septoria Tritici, Desm., produces irregular brown spots on the leaves of wheat, and is occasionally harmful. *S. nodorum*, Berkeley, (= *S. glumarum*, Pass.) forms blotches on the glumes of wheat.

Septoria Gladioli, Passer   Hard Rot of Gladiolus.

This fungus causes serious injury to the corms and foliage of gladiolus in England, Germany, and the United States. It has been investigated by Massey [29]. Pale areas, which become brown, appear on the leaves, and spores formed in these are washed down to the soil and infect the corms, producing dark-coloured lesions which remain hard. Pycnidia are rarely formed on the corms, and the disease is probably not disseminated during storage. The fungus may, however, persist in the soil for at least four years. When the corms are lifted only healthy ones should be kept, and these should be planted in soil free from the fungus. The spores are usually 3-septate, almost straight, and measure 20-$55 \times 2 \cdot 25$-4μ.

Septoria Azaleae, Voglino   Leaf Scorch of Azalea.

Salmon and Ware [10] have recently described an outbreak of this disease on azaleas imported from Belgium. The fungus produces brown areas on the margins and tips of the leaves, which extend

and lead to defoliation. The spores are 1–4 septate, oblong-linear with rounded ends, and measure $11–34 \times 1\cdot5–2\cdot5\,\mu$.

Dilophospora, Desm.

Pycnidia globose, black, ostiolate ; spores cylindrical, unicellular, hyaline, with hair-like appendages at both ends.

Dilophospora Alopecuri, (Fr.) Fr. (= *D. graminis*, Desm.)

Pycnidia 120–300 μ in diameter ; the spores (without appendages) measure $8\cdot5–16 \times 1\cdot6–2\cdot5\,\mu$.

This fungus, in association with the nematode worm, *Tylenchus Tritici*, causes a disease ('Twist') of wheat and rye, although it is uncommon in England. According to Atanasoff[31] the relation between the fungus and the nematode is an obligate one, the latter introducing the spores into the host plant. The nematode, however, is sometimes present alone, producing the disease of the grain known as 'Ear Cockles'.

The symptoms of the Dilophospora disease first appear on the third or fourth leaf as small pale spots, in the centre of which black pycnidia develop rapidly. The infected leaves usually soon die. The leaf sheaths then show similar lesions, which may cause the deformed ears to emerge incompletely. Some affected plants die outright. When the plants are attacked later in their growth the ears emerge, but are more or less affected by the fungus and the eelworm.

The eelworm galls on partly infected ears become overgrown by the fungus, and if sown with the grain are a source of infection of both diseases. The spores, adherent by means of the appendages, are carried by the nematodes by way of the leaf sheaths to the growing points of the seedlings, whence infection by the fungus occurs. Secondary spores of a similar type may be formed by the fungus after introduction into the seedling. Owing to movement of the eelworms within the young plants the fungus is carried about, so that successive leaves become infected. According to Atanasoff[31] infection is dependent upon only a slight admixture of the fungus spores with the nematodes, a heavy contamination of nematodes by

the fungus resulting in the host plant remaining free from both diseases.

If the grain is sown free from admixture with the eelworm, the fungus disease is automatically eliminated. Grain contaminated with 'Ear Cockles' can be freed by pouring it into a 20 per cent. solution of common salt. On stirring vigorously, the nematode galls come to the surface and can be skimmed off. The sound grain should then be rinsed in water and dried immediately. Infected straw should not be used for bedding animals as the disease may be spread in the manure. Risk of infection by nematodes which remain alive in the soil is obviated by crop rotation.

Leptothyrium, Kunze and Schweinitz

Pycnidia dimidiate, scutiform, black; conidia oblong or fusoid, unicellular, hyaline.

Leptothyrium Pomi, (Mont. and Fr.) Sacc. Fly Speck of Apple.

Apples are sometimes dotted over with minute black specks, which have been referred to this species. The fungus is entirely superficial; the specks are usually sclerotia, but they may develop into pycnidia.

Gloeodes, Colby

As in Leptothyrium, but the pycnidium is gelatinous within.

Gloeodes pomigena, Colby   Sooty Blotch of Apple and Pear.

'Sooty Blotch' and 'Fly Speck' were formerly considered to be two phases of the same disease, but Colby[32] has shown that they are caused by different fungi. The superficial mycelium forms irregular black blotches on apples and pears, the mycelium being sometimes fern-like in appearance. Pycnidia rarely occur. The spores are oblong, sometimes slightly curved, unicellular, hyaline, $10-20 \times 4-7 \mu$. Chlamydospores may be formed in the mycelium.

'Sooty Blotch' is most prevalent after heavy rain in the late summer. It does not spread appreciably in storage.

A form of 'Sooty Blotch' occurs fairly commonly on apples in England, but it is not known whether this is caused by the same fungus as that described by Colby in the United States.

Heteropatella, Fuckel

Pycnidia basin-shaped, brownish-black, opening by tooth-like segments; spores fusoid or falcate, attenuated at both ends, unicellular to 3-septate, hyaline.

Heteropatella Antirrhini, Buddin and Wakefield

Pycnidia 500–600 μ in diameter; spores 2–3 septate, without appendages 25–30 × 3–4 μ, apical appendage 20–25 μ, basal appendage up to 10 μ long. Pycnidia are found only on over-wintered dead stems.

The Hyphomycete stage (*Cercosporella Antirrhini*, Wakefield), with similar spores, occurs commonly as a parasite during the summer.

According to Buddin and Wakefield [33] this fungus causes the commonest and most destructive disease of Antirrhinums in England. It attacks vigorous leaves and stems, producing sunken patches with a water-soaked appearance. The diseased areas become brown later, and entire shoots may appear as if scorched. The diseased parts of the leaves sometimes fall away, giving a 'shot-hole' effect. The disease is most prevalent during cool, moist weather. Varieties of Antirrhinums with red pigment in the shoots appear to be comparatively resistant to the disease.

Rhizosphaera, Mangin and Hariot

Pycnidia ovoid to spherical, black, rupturing irregularly at the apex, formed over the stomata; spores unicellular, ovoid, hyaline.

Rhizosphaera Kalkhoffii, Bubák, attacks the leaves of conifers in Great Britain and Ireland, especially *Picea pungens* var. *argentea*, and *P. sitchensis*, the former of which it may defoliate and kill, as described by Wilson and Waldie [34]. Affected needles first become pale and then purplish-brown. The pycnidia are 80–150 μ in diameter, and the spores measure 7–10 × 3–4 μ.

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FUNGUS DISEASES (*continued*): MELANCONIALES

MELANCONIALES

THE conidiophores of this group are densely aggregated in pustules (acervuli), but are not enclosed in pycnidia. Here also, many forms have been shown in recent years to be conidial stages of ascomycetous fungi.

The genera are often ill-defined. For example, there is no adequate distinction between Gloeosporium and Colletotrichum.

Gloeosporium, Desm. and Mont.

Pustules (acervuli) pale or fuscous; spores ovate or oblong; conidiophores short, needle-shaped. The spores on germination often become uniseptate.

Many species of Gloeosporium have been found to be conidial stages of Glomerella, Gnomonia, and other Ascomycetes.

Gloeosporium ampelophagum, (Pass.) Sacc. (= *Sphaceloma ampelinum*, de Bary) Grape Anthracnose.

Grape anthracnose occurs on the berries, leaves, and young stems wherever the vine is cultivated. Upon the berries ashgrey spots appear, which enlarge and become sunken; between the pale central part and the brown border a reddish band is apparent. The spores are elliptical or oblong, and measure $5-6 \times 2 \cdot 5 - 3 \cdot 5 \mu$. The disease is difficult to control, but good results are often obtained by spraying the dormant vines with 10 per cent. sulphuric acid, followed by applications of Bordeaux or Burgundy mixture during spring.

Gloeosporium caulivorum, Kirchner (= *Kabatiella caulivora*, (Kirch.) Karakulin) Clover Scorch.

This species has been placed in the genus Kabatiella by Karakulin [1], a genus characterized by a pseudobasidial type of fructification, in which the conidiophores bear several conidia at the end, or rarely the sides of the swollen part; the conidia may give rise to other spores by budding.

This fungus produces sunken, dark streaks and spots on the stems and stalks of red clover. The disease may cause considerable losses of fodder in England and other countries, and a badly affected field may present the appearance of having been scorched. Ware [1a] points out that various strains of one and the same species of clover may show great differences in susceptibility. The spores are curved and measure 10–30 × 4–5 μ.

Sampson [1b] has compared this fungus with other species of Gloeosporium and Colletotrichum which also cause anthracnoses of red clover and related plants.

Gloeosporium limetticolum, Clausen   Withertip of Limes.

In Dominica a very serious disease of limes is caused by this fungus, which threatens to destroy the crop in some parts of the island. A report on the disease was made by Wiltshire [2] in 1925. The fungus causes a die-back of the young shoots and the development of brown blotches in the leaves, which fall rapidly. The flowers also are affected, and frequently no fruit is set. Fruit which is attacked late may reach maturity, but is greatly deformed. The disease is most prevalent in inland valleys where the annual rainfall reaches 250 inches. On the drier coastal belt, where the rainfall is only 50 inches, the disease is not so severe. Until resistant varieties have been found, it is likely that limes will only be grown successfully in the drier districts of Dominica.

According to Cunningham [3] the fungus is also probably responsible for a grey scab of citrus fruits in New Zealand.

Gloeosporium alborubrum, Petch, attacks young stems, leaves, and fruits of Para rubber (*Hevea brasiliensis*) in Malaya and Ceylon. Twigs are generally infected some distance below the apex. Shoots killed by this fungus should be cut out in young plantations, as *Botryodiplodia Theobromae* often follows it. Leaves are most liable to attack soon after unfolding, and the fungus may occasionally cause considerable defoliation. The pustules are pink, and the spores measure 15–20 × 3–4 μ. Other species of Gloeosporium and Colletotrichum also occur on rubber leaves.

Gloeosporium album, Osterw., causes a storage rot of apples and pears in Germany as described by Osterwalder [3a], and in England as recorded by Kidd and Beaumont [3b]. The rot usually begins

around the lenticels ; the affected tissues are brown and sunken, and bear white acervuli in concentric zones. The spores are generally slightly curved, and are longer and narrower than in *G. fructigenum* (see p. 200).

COLLETOTRICHUM, Corda

Characters as in Gloeosporium, but the conidiophores are interspersed with long, black setae. The setae, however, are somewhat irregular in occurrence, and it would be more satisfactory if Gloeosporium and Colletotrichum were fused into a single genus. Some 'species' of Colletotrichum are now considered to be conidial stages of *Glomerella cingulata*.

Colletotrichum lindemuthianum, (Sacc. and Magn.) Bri. and Cav. Anthracnose of Dwarf and Runner Beans.

Conidia oblong, ends rounded, straight or curved, $15–19 \times 3\cdot5–5\cdot5\,\mu$; the setae are very irregular in occurrence.

This fungus possibly belongs to *Glomerella cingulata* (see p. 200), or to a closely related species of Glomerella.

Considerable losses are sometimes caused by this anthracnose,
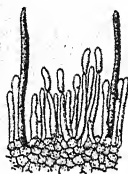
FIG. 59. Section of acervulus of *Colletotrichum lindemuthianum* showing spores and setae. × 175. (*R. W. Marsh.*)

which attacks the stems, leaves, and pods of dwarf and runner beans, forming dark, sunken spots on which pink acervuli are borne. The mycelium may penetrate the pods, and reach the seed, hence the disease is sometimes seed-borne. The disease is most prevalent during cold, wet weather, but is somewhat uncommon in England. Whetzel[4] indicates that careful seed selection, coupled with the use of the more resistant varieties, is the best safeguard against this disease. The elaboration of new resistant varieties in the United States is complicated by the occurrence there of several distinct biologic forms of the fungus, as described by Leach[5].

Dey[6] has studied the initiation of infection by this fungus : the germ tube on coming into contact with the host forms an appressorium, from which a peg-like 'infection hypha' grows out and ruptures the cuticle mechanically. Leach[5] indicates

that in resistant varieties of beans the fungus effects penetration, but the mycelium soon becomes disintegrated together with the protoplasts of the host cells in immediate association. Schaffnit and Böning[7] record the occurrence of pycnidia in pure cultures.

Colletotrichum oligochaetum, Cav.   Cucumber Anthracnose.

Bewley[8] points out that this fungus is sometimes troublesome in cucumber cultivation under glass.   The disease attacks the leaves, stems, and fruit, and may appear at any time during the growth of the plant.   The lesions on the leaves commence as pale water-soaked areas, but these soon become dry and brown.   The spots rapidly coalesce, and the leaf dies.   The lesions on the stems may kill the plant outright.   On the fruits the fungus produces depressed areas, which, owing to abundant spore formation, become pink in colour, and ultimately black.   The progress of the disease is most rapid at high humidities.   After a severe outbreak of the disease the fungus may continue to live saprophytically in the greenhouse on old woodwork and straw debris; the water supply also may be a source of contamination.

After a severe attack, Bewley[8] advises that the greenhouse be thoroughly disinfected during the dormant season with an emulsion of cresylic acid.   When the disease first appears, the affected leaves should be burnt and the plants sprayed at intervals with lime-sulphur of spec. grav. 1·005.   As cucumber leaves are difficult to wet, a small amount of flour, 5 lbs. to 100 gallons spray fluid, should be added to act as a 'spreader'.

Colletotrichum atramentarium, (Berk. and Br.) Taub. (= *Vermicularia varians*, Ducomet)   Black Dot of Potatoes.

There has been much confusion concerning the nomenclature of this fungus, but this has now been cleared up by Dickson[9]. *C. tabificum*, (Hall. p.p.) Pethybr., is the same organism.   The fungus attacks the underground parts of potatoes and tomatoes.   According to Crépin[10] a very serious disease of potatoes is caused by it in France, the symptoms being rolling of the upper leaves, yellowing, and premature death of the foliage.

A diseased potato plant shows a dry rot of the stolons, tubers, and roots. Black sclerotia appear on or near the surface of the lower part of the stem and underground parts. These are often provided with typical Colletotrichum setae. Spore-bearing pustules also occur on the diseased tissues. In England the black sclerotia of this fungus are frequently found on dead haulms and sometimes on the tubers, but the fungus appears to be practically harmless to potatoes under English conditions.

C. atramentarium, however, is responsible for a serious disease of tomatoes grown under glass in England, as described by Bewley [11]. The fungus attacks the roots and base of the stem, causing death. The outer tissues of the roots readily come away from the wood, and innumerable small sclerotia are found embedded both in the outer tissues and the wood. Sometimes the root system is not wholly destroyed and the plants bear a diminished crop. Greenhouse soil contaminated by this fungus should be sterilized by steam.

Colletotrichum phomoides, (Sacc.) Ches.   Tomato Anthracnose.

This ripe-rot of tomatoes, although widespread in the United States, is rarely serious. Brooks and Searle [12] have recorded its occurrence in England. The parasite enters the ripening fruits through wounds, which are usually minute. The young lesions are circular in outline, but they may later involve the whole fruit, which is reduced to a pulpy mass.

Colletotrichum gloeosporioides, Penzig

There has been much dispute as to whether this is a serious parasite of citrus trees or merely a saprophyte. It is certainly far less harmful than is *Gloeosporium limetticolum* to limes, but it is probably capable, under some conditions, of causing spotting of the leaves and blotching of the fruits. It causes a stem-end rot of oranges and grape-fruit in storage. The pustules are frequently devoid of setae, and, in this condition, are difficult to distinguish from *G. limetticolum. C. gloeo-sporioides* probably belongs to *Glomerella cingulata,* (Stonem.) Spauld. and v. Sch.

Small [13] points out that *C. coffeanum,* Noack, which causes

a brown blight of coffee leaves and ripening berries, and occurs on several other cultivated plants in Uganda, is probably identical with *C. gloeosporioides*. Several strains of Small's *C. coffeanum* gave rise to *Glomerella cingulata* in culture. McDonald [14] reports that a serious anthracnose of green coffee berries in Kenya Colony is caused by another strain of *C. coffeanum*, Noack.

Colletotrichum circinans, Vogl.   Onion Smudge.

This disease occurs on onions, shallots, and leeks, but is most common on white onions. It has been investigated by Walker [15]. Infection occurs from the soil and may occasionally cause 'damping off'. More frequently, however, the fungus attacks the neck and bulb shortly before harvest, causing the appearance of black smudges. The stromata are often arranged in concentric circles. When the attack is severe the fungus grows through the outer dry scales into the inner fleshy ones, causing considerable shrinkage. The disease sometimes develops in storage, but in such cases initial contamination probably occurred in the soil.

Walker [16] points out that, in the United States, coloured onions are practically immune from attack by this fungus, and attributes this resistance to the toxic influence of the pigments, or bodies associated with them.

C. Lini, (Westerd.) Toch. (= *Colletotrichum linicolum*, Pethy. and Lafferty)   Seedling Blight of Flax.

Pethybridge and Lafferty [17] state that this species is often destructive to flax seedlings in Ireland. The disease is carried over by mycelium in the seed coat, which infects the cotyledons on germination. Spores produced on the cotyledons in turn infect the young stems, which often collapse in consequence. When the stem attack is mild, the plant may recover, but the leaves and fruits of older plants may also be attacked. The mycelium in the fruit often invades the coats of the seeds. Acervuli are produced chiefly in the lesions on the leaves and stems. The fungus has been found on seed from Russia, Holland, Canada, the United States, and Japan.

Y

Polyspora, Lafferty

Acervuli minute, gelatinous, hyaline or milky, formed directly over the stomata ; conidia unicellular with bluntly pointed ends, of varying shape (oval, cylindrical, &c.), hyaline.

Polyspora Lini, Lafferty   Browning and Stem-Break of Flax.

Conidia as described for the genus, $9–20 \times 4 \mu$. Conidia sometimes occur below the epidermis as well as in acervuli above the stomata. In culture, conidia may be formed in groups from any cell of the mycelium, and the conidia may bud to produce others. Karakulin [1] suggests that this fungus may perhaps belong to the genus Kabatiella.

This disease was first investigated by Lafferty [18] in Ireland. It has also been reported in the United States, Russia, and East Africa, and it probably occurs wherever flax is cultivated. The 'browning' phase of the disease first becomes conspicuous just before the crop is 'pulled', appearing as groups of 'browned' plants in isolated patches, which extend rapidly until the whole field may be involved. The 'browning' of the plants is due to the presence of innumerable diseased areas on the fruits, sepals, leaves, and stems. When the stem is affected at an earlier stage it may bend over or be completely broken near the first node, hence the name 'stem-break'. The mycelium permeates only the parenchymatous tissues. Acervuli are produced in large numbers over the stomata. From an affected fruit the mycelium proceeds to invade the outer layers of the seed coats, but the embryo remains uninjured. Infected seeds often produce conidia at the hilum.

The disease is transmitted primarily by the use of infected seed, and such seed has been received from many countries. Upon germination, the seed coat is carried up on the cotyledons, which often become infected. The conidia produced on the cotyledons infect other parts of the growing crop, the disease spreading most rapidly in warm, damp weather. Infected seed can be disinfected by treatment with a 0.59 per cent. solution of formaldehyde, but, as the treatment reduces germination somewhat, such seed should be sown thicker than usual. It is best, however, to obtain seed if possible from a crop free from

' browning '. Owing to this disease the production of healthy flax seed for sowing is almost impossible in a climate like that of Northern Ireland.

Myxosporium, Link

This genus is essentially the same as Gloeosporium, but the pustules are formed on twigs instead of chiefly on leaves and fruits.

Myxosporium corticola, Edgert. Surface Canker of Apple and Pear.

Surface canker of apple and pear trees is characterized by the occurrence of irregular, slightly depressed areas on the trunk and larger limbs, which are sharply delimited from the healthy tissue by cracks. Only the outer layers of the bark are killed, the progress of the fungus inwards being checked by the formation of a cork layer. The disease, which occurs commonly on adult apple trees in England, is practically innocuous. The spores of the fungus measure $18\text{--}36 \times 6\text{--}9\,\mu$.

Marssonina, Fisch.

Pustules long covered by the epidermis, pale; spores ovate or elongated, hyaline, uniseptate.
The name was formerly spelt Marssonia.

Marssonina panattoniana, Berl. Ring Spot of Lettuce.

' Ring-spot' of lettuce is a serious disease both under glass and in the open, especially in the latter case in crops harvested in the spring. It has only recently become a serious trouble in lettuce cultivation in England, an epidemic of it having been first reported by Salmon and Wormald [19] in 1923. The disease also occurs on the Continent and in the United States.

The diseased areas on the leaf appear first as brown, water-soaked spots, which soon become white with the spores of the fungus; the central portions of the spots then fall away, leaving holes with a white margin. The under side of the midrib is also often attacked, the spots here becoming elongated, sunken brown patches. The spores measure $15 \times 3\,\mu$.

The disease starts on the outer leaves and spreads inwards to the ' heart ', when the whole plant may die. There is

evidence that the disease is perpetuated from the remains of the previous lettuce crop, so a system of rotation should be practised. At the same time manure containing lettuce debris should not be used for this crop.

Marssonina salicicola, Bresad., or a closely related species has been found by Nattrass * to cause small lesions or cankers in the stems of *Salix purpurea*. The spores measure 15–17×6–$8\,\mu$. Murray [20] records it as causing a leaf spot and twig canker of *S. babylonica* and *S. fragilis* in New Zealand.

Marssonina Fragariae (Sacc.) Kleb., causes a leaf scorch of strawberries. The perfect stage is *Diplocarpon earlians*, (Ell. & Everh.) Wolf.

Septogloeum, Sacc.

Pustules very small, pallid ; spores long, 2 or more septate, hyaline.

Septogloeum arachidis, Rac.

Between 1894 and 1902 the cultivation of ground nuts on a large scale in Bombay was rendered almost impossible by this fungus, but owing to the introduction of more resistant varieties from other countries the growing of this crop has been resuscitated. The disease appears first in the form of dark spots on the leaves, and the affected leaves then soon fall. The spores are formed chiefly on the under surface of the leaves; they are divided by cross septa into 3–8 cells, and measure 20–55×6–$8\,\mu$.

Pseudodiscosia, Höstermann and Laubert

Conidial pustules formed under the cuticle, which on being ruptured surrounds the pustule like a collar ; conidia spindle-shaped, often slightly curved, tapering to appendages at both ends, usually 2–3 septate, hyaline.

Pseudodiscosia Dianthi, Höst. and Laub.　　Carnation Leaf Rot.

This fungus was first described by Höstermann and Laubert [21] as the cause of a serious disease of carnations in Germany. It has since been recorded by Salmon and Ware [22] in England, and it occurs also in Holland.

The disease is characterized by a brown rot of the leaves, which may be wet or dry according to the weather. The base of the leaf

* Information kindly given by Dr. R. M. Nattrass, Long Ashton, Bristol, in advance of publication.

is the part most frequently attacked, and young leaves are the most susceptible. The German authors state that the fungus also attacks the stems, flower stalks, bracts, and sepals. The conidial pustules appear as minute grey spots. The spores measure 12–42×3–$7\,\mu$, but they are often devoid of appendages, when they are 12–$24\,\mu$ long. Low temperatures and wet weather appear to favour the disease.

Pestalozzia, de Not.

Pustules black; spores long, 2 or more septate, brown, the end cells sometimes hyaline, with one or more hyaline apical appendages.

Pestalozzia Theae, Sawada   Grey Blight of Tea.

When first discovered it was thought that this fungus might cause serious damage to tea, but, according to Petch [23], it is rarely important except when it attacks young foliage or plants weakened by faulty conditions of cultivation. The characteristic spot of 'Grey Blight' is irregularly circular, and varies greatly in size. When old, the spot becomes greyish. The pustules arise on the spots as minute black points; the spores measure 20–35×5–$11\,\mu$. The fungus also occurs on the young stems and may kill them back for a short distance. This species grows commonly as a saprophyte on dead tea shoots.

Pestalozzia palmarum, Cooke

This fungus causes a brownish spotting of the leaves of coconut and other palms, but it is harmless except when young plants are attacked. If need be, the disease can be controlled by spraying the young plants with Bordeaux mixture. The spores of this species are smaller than those of *P. Theae,* measuring 15–21×5–$6\,\mu$.

Pestalozzia Hartigii, Tubeuf, attacks the bark of young conifers just above soil level, and kills them. The region affected shrinks, but, just above this, the stem is swollen. In Britain the disease is sometimes seen on conifers imported from the Continent.

Cylindrosporium, Unger

Pustules white or pallid; conidia filiform, straight or curved, unicellular, hyaline.

Cylindrosporium padi, Karst.

This is the commonest cause of the 'shot-hole' disease of plum and cherry leaves in the United States, and it may also be a cause of this trouble in England. The fungus produces small, brown areas on the leaves, more or less circular in outline, which after a time fall away from the lamina. The pustules of the fungus are only rarely found on the portions which have fallen away, but these occur more frequently on certain varieties in which the injured areas persist in the laminae. The spores are curved and measure $48-60 \times 2\,\mu$.

Cylindrosporium Chrysanthemi, E. & D., causes dark blotches on the leaves of chrysanthemums.

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FUNGUS DISEASES (*continued*) : HYPHOMYCETES,
MYCELIA STERILIA

HYPHOMYCETES

THE conidiophores of this group either remain more or less separate
from one another or are aggregated into coremia. It is sometimes
difficult to distinguish these fungi from the Melanconiales. Many
species (e. g. *Monilia fructigena*) have been transferred to the Asco-
mycetes in recent years.

Oospora, Wallr.

Fertile hyphae short, sparingly branched, slender ; conidia catenu-
late, globose to ovoid, hyaline.

Oospora pustulans, Owen and Wakef. Skin Spot of Potatoes.
The name 'Skin Spot' was first used by Pethybridge[1] to
describe a disease in which shallow, circular depressions, with
a slightly raised centre, or small raised areas are formed in
greater or less abundance on the surface of potato tubers. The
disease renders the tubers unsightly, and with seed tubers it
may be serious, as the eyes may be destroyed. 'Skin spot'
has been investigated by Owen[1a], who states that the fungus
penetrates a few layers of cells below the skin of the tuber,
being prevented from growing deeper by the formation of
a cork-barrier. The disease develops during storage, but it is not
known whether infection occurs from the soil. The fungus not
uncommonly kills the young shoots of tubers which have
sprouted in the clamps, such shoots becoming covered with the
white conidiophores of the fungus. The varieties which
appear to be most susceptible to 'Skin Spot' are 'Arran
Chief', 'King Edward', and 'British Queen'. Tubers which
are severely affected should not be used for seed.

Cephalosporium, Corda

Conidiophores short, upright ; conidia spherical or ovoid, hyaline
or lightly coloured, arising in small heads at the ends of the coni-
diophores.

Cephalosporium Costantinii, F. E. V. Smith

This fungus is the cause of a disease of cultivated mushrooms, which become spherical or invertedly pyriform, and dry and elastic, as described by Smith [2]. According to the same author, another species, *C. lamellaecola,* causes a 'mildewing' of mushroom gills, which is accompanied by fasciation.

Sporotrichum, Link

Conidiophores branched, not upright; conidia terminal or on short sterigmata, generally formed singly, ovoid or spherical, unicellular, hyaline.

Sporotrichum Citri, Butler (= *Sphaceloma Fawcetti,* Jenkins) Citrus Scab.

As pointed out by Doidge and Butler [3] this fungus was formerly wrongly diagnosed as *Cladosporium Citri,* Massee. Jenkins [4] considers it closely related to the fungus of grape anthracnose. The spores measure $2{-}6\cdot5 \times 1\cdot3{-}2\,\mu$.

'Citrus Scab' affects the leaves, young twigs, and fruit of the lemon, sour orange, and grape-fruit. It occurs in many citrus-growing countries. Only young tissues can be infected. On the leaves the scabs, variously coloured when young, are found chiefly on the under surface; affected leaves may be considerably distorted. On the fruit the disease occurs as small corky projections, and in lemons it is accompanied by distortion. Fawcett [5] states that infection takes place only between 61° and 73° F. (16° and 23° C.).

The disease is best controlled by spraying the trees, just before and just after flowering, with Bordeaux mixture to which 1 per cent. paraffin emulsion has been added (see p. 363).

Verticillium, Nees

Conidiophores upright, branched, the lateral branches being arranged in whorls; conidia arising singly or in groups, soon falling away, spherical or ovate, unicellular, hyaline or slightly coloured.

Verticillium albo-atrum, Reinke and Berth.

Conidia ovate-oblong, hyaline, $5\cdot5 \times 2\cdot7\,\mu$ (average).

This fungus causes a wilt or desiccation of a great number of woody and herbaceous plants, including cherry, tomato, cucumber, potato, and hop. The diseases induced by this fungus have been investigated by Pethybridge [6], Bewley [7], van der Meer [8], and others. The fungus usually first infects the young, uninjured roots from the soil, but in the potato it is often carried over in the vascular tissue of the seed tubers. The mycelium is chiefly found in the vessels of the roots and stems, and its presence there is associated with the formation of a gummy substance which causes a yellowish-brown discoloration of the vascular strands. Most plants exhibit a wilt when badly invaded by this fungus, which, according to Bewley [7], is due primarily to the secretion of a toxic substance that is carried up in the transpiration stream. The wilt of tomato plants affected by this disease is so pronounced as to give the name ' Sleepy Disease ' to the affection. With potatoes, on the other hand, Pethybridge [6] points out that the commonest effect of the fungus is to cause a dwarfing of the plant, associated with a rolling upwards of the leaflets and accompanied later by a gradual desiccation of the shoots.

' Sleepy Disease ' caused by this fungus * is of common occurrence in tomato cultivation under glass. Bewley [7] states that the fungus is most active at temperatures of 21°–23° C. (70°–73° F.), but that it rarely causes infection above 25° C. (77° F.). Wilted plants can usually be induced to recover by raising the temperature above 25° C. (77° F.). Tomato plants which are checked in their growth when young are particularly liable to attack. Most varieties of tomatoes grown in England are susceptible, but ' Manx Marvel ' and ' Bides Recruit ' are very resistant. It is advisable to partially sterilize the soil of tomato houses in which the disease has been prevalent, as described by Bewley [8a].

The disease is not very common in potatoes in Britain. Infected plants generally give rise to diseased tubers. The fungus does not cause a rot of the tuber, but, entering at the

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* It seems likely that the fungus described by Bewley [7] was really *V. Dahliae*, Kleb., a species very closely related to *V. albo-atrum*. Van der Meer [8] states that both species cause a wilt of tomato plants.

heel end, causes a brown discoloration of the vascular ring. Such discoloration may also be brought about by other causes. As a general rule tubers which show a brown discoloration near the heel end when cut across should not be used for 'seed' purposes.

Verticillium Dahliae, Klebh.   Blue Stripe Wilt of Raspberry.

This species differs from *V. albo-atrum* in having slightly smaller spores and in producing micro-sclerotia abundantly in culture.

'Blue Stripe Wilt' of raspberry has been described by Harris[9], who considers that the causative fungus is *V. Dahliae* and that it may be identical with *Acrostalagmus caulophagus*, stated by Lawrence[10] to cause 'Blue Stem' disease of the black raspberry in the United States.

During the summer the lower leaves of newly infected canes show a yellow or reddish brown discoloration of the lamina between the main veins, but only a part of the leaf may be thus affected. These variegated leaves fall prematurely. A little later the lower part of such a cane shows a dark blue or bluish-brown discoloration, and the cane often wilts. Fruiting canes may be killed in the winter, or they may put forth leaves in the spring, which soon become yellow and wither. The fungus infects the root-stock and grows upwards in the stems.

In Britain the varieties 'Bath's Perfection' and 'Red Antwerp B' are very susceptible to this disease. Raspberry canes should be propagated only from healthy stools.

V. Dahliae causes a wilt of many other plants, including tomato, lilac, Daphne, black currant, dahlia, phlox, and China aster, as pointed out by van der Meer[8].

Verticillium Vilmorinii, Westerdijk & v. Luijk (= *Acrostalagmus Vilmorinii*, Guég. = *Cephalosporium Asteris*, Dowson)

A serious disease of Michaelmas Daisies has been found to be due to this fungus by Guéguen[11] and Dowson[12]. Westerdijk and van Luijk[13] point out that the organism properly belongs to the genus Verticillium, although conidiophores of the Cephalosporium type are formed abundantly in culture, as

described by Dowson [12]. The conidia on the average measure
$4 \times 1.5 \mu$.

The first symptom of disease is a yellow mottling of some
of the lower leaves in the early summer, followed by complete
yellowing and browning. In some cases the entire shoot wilts
as the summer advances. According to Dowson [12] the fungus
invades the root-stock from the soil through abrasions, perhaps
caused by insects, and, entering the wood vessels, passes
upwards into the stems. A toxin is produced by the growing
fungus, which, carried upwards in the transpiration current,
causes mottling of the leaves some distance beyond the
mycelium.

Diseased plants produce suckers at an early stage, and the
fungus often passes into these, so that unless care is taken the
disease is liable to be propagated with the suckers. Dowson [12]
points out that healthy plants may be obtained by using as
cuttings the upper parts of suckers into which the fungus has
not grown.

Trichothecium, Link

Conidiophores upright, unbranched, septate; conidia terminal,
forming a small head, oblong to pyriform, 2-celled, hyaline.

Trichothecium roseum, Link (= *Cephalothecium roseum*, Corda)

This pink mould is one of the commonest of saprophytes, but
it occasionally causes a fruit rot of apples and pears, following
in the wake of the scab fungus as the fruits approach maturity.
Very rarely also, when apples are stored under humid con-
ditions, the fungus may begin to develop on the ' eye ' and
pass into the core, from which a rot is set up inside the fruit.

Mycogone, Link

Conidiophores short, lateral; conidia (chlamydospores) formed
singly, unequally 2-celled, the upper cell larger, echinulate.

Mycogone perniciosa, Mag.   White Mould of Mushrooms.

Spores of two kinds occur: (*a*) thin-walled, 1-septate conidia borne
terminally on Verticillium-like conidiophores, hyaline, $15–20 \times
3–4 \mu$. (*b*) Chlamydospores formed singly on short branches, uni-
septate: upper cell spherical with a thick, warted wall, light

brown, $18\text{-}20 \times 14\text{-}17\,\mu$; lower cell thin-walled, hyaline, $10\text{-}14 \times 9\text{-}12\,\mu$, incapable of germination.

This fungus frequently attacks cultivated mushrooms and sometimes does great damage. The fungus may appear as a dense mould on the stipe or gills of normally formed mushrooms, or it may cause great deformity, characterized by a greatly enlarged stipe and much reduced pileus and gills. In extreme types of the latter affection the mushroom is said to be 'sclerodermoid' and shows none of the parasite externally. Smith [2] states that the sclerodermoid forms arise when the mushrooms are affected at a very early stage. According to him each fructification is infected separately, the mycelium being rarely attacked. Once infected, the mushroom quickly decomposes owing to enzymic action on the part of the parasite.

Mycogone perniciosa grows saprophytically in the manure and soil of which mushroom beds are composed, so that, once a 'house' is infected seriously, the compost should be removed and the interior of the house disinfected with 2 per cent. formalin. Disinfection with formalin also destroys the mushroom fly which disseminates the spores of the parasite. If only a small part of the bed is attacked, it can be sterilized *in situ* by soaking it with formalin. The parasite is sometimes present in the 'spawn' used to inoculate the beds, so that care should be taken to obtain it only from places where the parasite is known not to exist.

Rhynchosporium, Heinsen

Conidia curved, beaked, with a nearly median septum, hyaline.

Rhynchosporium Secalis, (Oud.) Davis   Leaf Blotch of Barley and Rye.

This fungus affects the leaves of barley, rye, and certain grasses, e.g. *Bromus mollis*, producing spots or blotches on any part of the lamina. The spots or blotches first appear as water-soaked areas of irregular shape; later, they become greyish with a dark brown margin, and increase considerably in size, so that sometimes the greater part of the lamina is

affected. In barley, infection often begins at the auricles. The spores are formed over practically the whole surface of the blotch, and measure $11–16 \times 3·5–5 \, \mu$.

FIG. 60. 'Leaf blotch' of barley caused by *Rhynchosporium Secalis*. $\times \frac{1}{2}$.

Although the fungus is quite common on English barley in some seasons, especially in late winter and early spring, it does not appear to cause appreciable harm to the crop.

Ramularia, Unger

Conidiophores fasciculate, simple or with short branchlets ; conidia ovate-cylindrical, sometimes catenulate, usually 1–several times septate, hyaline.

Ramularia Armoraciae, Fckl.

White, papery spots are produced on the leaves of horse-radish by this fungus. The diseased areas often fall away, giving a 'shot-hole' effect. Small sclerotia are formed in the diseased

tissues in the autumn ; these are a means of over-wintering, as they give rise to conidia in the spring.

Ramularia Vallisumbrosae, Cav.

This fungus affects the leaves and stalks of Narcissus, producing pale yellow streaks ; it has recently done considerable damage in the Scilly Isles. Small spherical or irregular black bodies, probably sclerotia, are produced in the leaves, as well as conidia.

Ramularia lactea, (Desm.) Sacc., occurs on violets, causing irregular, white spots on the leaves.

Cercosporella, Sacc.

Conidiophores simple or branched ; conidia worm-like, many-septate, hyaline.

Cercosporella Pastinacae, Karst., causes a leaf spot of parsnips.

Thielaviopsis, Went.

Conidiophores simple, septate ; conidia of two kinds ; macroconidia catenulate, ovate, fuscous ; microconidia catenulate, cylindrical hyaline, arising from within a specialized conidiophore.

Thielaviopsis paradoxa, (De Seynes) v. Höhn. (= *T. ethaceticus,* Went.)

Macroconidia 16–19×10–$12\,\mu$; microconidia 10–15×3.5–$5\,\mu$.

This fungus is widely distributed in the tropics and attacks sugar-cane, the coconut palm, banana suckers, and pineapples. The extremities of sugar-cane cuttings are often affected by it, especially in dry weather, and, more rarely, standing cane is affected through leaf scars or after attack by boring beetles. The fungus causes a black rot in the centre of the cane, producing a smell resembling that of a ripe pineapple. Attack on the cuttings can usually be prevented by dipping the extremities in Bordeaux mixture before planting. Petch [14] states that in Ceylon the fungus affects the stems of coconut palms, causing the emergence of a viscid fluid ('stem-bleeding disease'), but it is rarely harmful. Young pineapple cuttings may be invaded by the fungus through the end in the soil. During storage and in transit pineapple fruits are often rotted

by it, especially if kept in a warm, humid atmosphere ; the fungus does not grow below 10° C.

Fusicladium, Bon.

Conidiophores short, olivaceous ; conidia terminal, ovate, at first unicellular, then often uniseptate, brownish.

Fusicladium Cerasi, (Rab.) Sacc. (= *Cladosporium carpophilum,* Thüm.)*   Peach and Cherry Scab.

Peach Scab occurs wherever peaches and nectarines are grown on a large scale. The fungus has also been recorded on cherries, plums, and apricots. On the peach fruit the disease first appears as small, irregular spots, which enlarge, darken, and frequently coalesce. In severe attacks the fruits may be badly scabbed or cracked, and gum often exudes from the cracks. Badly affected fruits sometimes fall prematurely. The fungus grows in the skin of the fruit, from which dark conidiophores arise, giving the lesions an olivaceous colour. In peaches the fungus also produces spots on the leaves and young stems, over-wintering in the latter, as described by Keitt [14a].

The disease can be controlled by summer spraying with lime-sulphur of specific gravity 1·002.

Cladosporium, Link

Conidiophores arising in tufts, slightly branched, olivaceous ; conidia ovoid, at first non-septate, then often 1–2 septate, slightly coloured, formed sympodially at the ends of the conidiophores.

Cladosporium herbarum, Link

The conidia are very variable in size ; they may be unicellular or 1–2 septate. Under humid conditions the conidia bud forth and produce chains of small, globose spores, which were formerly considered to be a distinct fungus, *Hormodendron cladosporioides,* Sacc. Brooks and Hansford [15] have indicated that this species is very polymorphic. According to Jancewski [16] the perithecial stage is *Mycosphaeralla Tulasnei,* Janc., but this has not been confirmed.

This ubiquitous mould is rarely parasitic. In a wet season

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* Bensaude and Keitt indicate (*Phytopath.,* vol. 18, p. 313, 1928) that the fungus of Cherry Scab (*F. Cerasi*) may be distinct from that of Peach and Plum Scab (*C. carpophilum*).

ears of wheat, especially after attack by *Erysiphe graminis*, may appear blackened in consequence of the growth of *C. herbarum*, but the grain is not usually damaged. The variety 'Benefactor' is not infrequently affected in this way in England. Bennett[16a] has studied the effect of *C. herbarum* on the growth of wheat in England, and has found that it is innocuous under normal conditions.

One of the many forms of this fungus causes a spotting of the twigs of the sweet orange, known as 'leprosis'. As the spots become older, they crack and coalesce, forming large patches of scaly and scabby bark.

Cladosporium album, Dowson

This species is essentially the same morphologically as the preceding, but is entirely hyaline. It is markedly parasitic on the foliage of sweet peas, both under glass and in the open, as described by Dowson[17]. Mildew-like patches appear on the leaves and wings of the stems, and these may spread so that the entire leaf surface is attacked. The tissues are gradually killed by the fungus, and severely affected leaves fall prematurely. The conidiophores emerge through the stomata and produce spores in chains as in the Hormodendron condition of *C. herbarum*. The fungus is only an active parasite at high temperatures. Under glass the disease can be controlled by ensuring adequate ventilation and by sulphuring.

Cladosporium cucumerinum, Ell. and Arth. Cucumber Gummosis.

In England this fungus commonly attacks the fruits and leaves of cucumbers under glass. It also occurs on pumpkins and muskmelons in the United States. Cucumber fruits are usually attacked late in the season. The disease is characterized by the appearance of small, sunken spots which spread rapidly and exude a gummy liquid. The lesions frequently crack and soon become covered with an olive-green, velvety growth of conidiophores. The fungus also forms light-brown, irregular spots on the leaves.

The disease is most severe at high temperatures and under

conditions of extreme humidity. It can be checked by efficient ventilation and by spraying with liver of sulphur mixed with a small quantity of flour paste.

Cladosporium fulvum, Cke. Tomato Leaf Mould.

Conidiophores densely crowded, olivaceous, septate, nodulose, bearing a few conidia near the apex ; conidia elliptic-oblong, uniseptate, almost hyaline, 10–20 × 4–6 μ.

'Leaf Mould' is probably the commonest disease of tomatoes grown under glass, occurring chiefly late in the season. The fungus appears on the under side of the leaves as an olivaceous mould, the corresponding parts of the upper surface being pallid. At a later stage the mould becomes purplish in colour, and the affected part of the leaf dies. The fungus spreads rapidly and is often epidemic. When the attack is severe, the foliage may be killed and the later part of the crop lost. In slight attacks only the older leaves are affected, and little harm is done. The fungus grows best at a temperature of 20° to 25° C. in a very moist atmosphere. The disease can be prevented from becoming severe by efficient ventilation and by keeping the atmosphere relatively dry. The variety 'Sterling Castle' is resistant.

FIG. 61. Conidiophores of *Cladosporium fulvum.* × 250. (*R. W. Marsh.*)

Clasterosporium, Schwein

Conidiophores short, dark ; conidia 2 or more septate, brownish.

Clasterosporium carpophilum, (Lév.) Aderhold (= *Coryneum Beyerinckii,* Oud.).

Conidiophores often grouped together ; conidia elongate-fusoid, obtuse, 3–5 septate, brownish, 23–62 × 12–18 μ.

This is a common cause of the 'shot-hole' effect in apricot, peach, almond, plum, and cherry leaves in Europe, as described by Aderhold[18]. Samuel[19] states that it is prevalent in Australia, and Cunningham[20] considers it a serious fungus pest of the

apricot and peach in New Zealand. The fungus is known in the United States under the name of *Coryneum Beyerinckii.*

Infection of the leaves results in the formation of circular, brown spots, which frequently fall away, leaving holes. According to Samuel[19] the cell walls on the margin of the spot become lignified, and the cells just beyond divide, the new walls becoming suberized; abscission occurs by the dissolution of the middle lamellae between the two layers of cells immediately outside the lignified zone. He states, however, that if moisture relations are unfavourable, abscission does not occur.

C. carpophilum also affects the fruit and young twigs, producing small scabs. This kind of attack is often accompanied by gumming. Cunningham[20] states that the fungus causes a die-back of the twigs of stone fruits in New Zealand.

The fungus over-winters in twig lesions and by means of spores entangled in the bud scales.

Heterosporium, Klotszch

Conidiophores arising in groups, often branched ; conidia oblong, 2–several times septate, granular or echinulate, pale brown.

Heterosporium echinulatum, (Berk.) Cke. Ring Spot of Carnations.

This fungus attacks the leaves of carnations and pinks, especially under glass. Pale circular spots are produced on the leaves, on which tufts of conidiophores arise in concentric zones. The conidia are 2–5 septate and measure 30–50 × 10–15 μ. Diseased leaves should be picked off the plants and burnt, and care should be taken not to water the leaves.

Heterosporium auriculi, Mass., occurs on cultivated Auriculas, *H. variabile*, Cke., on the leaves of spinach, and *H. Syringae* on lilac leaves.

Spondylocladium, Martius

Conidiophores simple, rigid ; conidia arising in whorls, fusoid, usually 2–septate, brownish.

Spondylocladium atrovirens, Harz Silver Scurf of Potatoes.

Conidiophores septate, brownish, up to 400 μ long; conidia elongate, ovate, apex narrowed, 5–7 septate, brownish, 30–50 × 6–9 μ.

This fungus commonly occurs on the surface of potato tubers in Britain, but is very rarely harmful. The mycelium is usually confined to the skin, from the surface of which the conidiophores arise. The diseased skin is often raised somewhat, so that the surface has a silvery appearance owing to the air space below.

Coniothecium, Corda

Mycelium very poorly developed; conidia gemmiform in origin, muriform and irregular in shape, brownish.

Coniothecium chomatosporum, Corda Rough Scab (Blister Disease) of Apples and Pears.

This disease of apples and pears occurs commonly in New Zealand and Australia, and it occasionally affects apples in England. Fruit affected by it is characterized by russetting and by the presence of irregular rough cracks. Cunningham[*] states that in New Zealand the fungus also affects the twigs and branches, producing small blisters in the bark. He has shown that a Phoma stage occurs in cultures. The disease can be controlled by cutting away the affected parts and by spraying the trees with Bordeaux mixture.

Macrosporium, Fries

Conidiophores fasciculate, more or less branched, dark-coloured; conidia apical, elongated or globose, muriform, dark-coloured.

It is sometimes difficult to distinguish this genus from Alternaria.

Some species of Macrosporium are known to be conidial stages of Pleospora. *M. parasiticum*, a weak parasite of onions, is considered to be the conidial stage of *P. herbarum*.

Macrosporium Solani, E. and M.

In the United States, Canada, and Bermuda this fungus attacks young potato and tomato foliage, the disease caused by it being known as 'Early Blight'. The fungus produces dark brown spots with concentric markings, which may extend rapidly under favourable conditions, although the disease is rarely epidemic. Weakened foliage is most liable to attack.

[*] Cunningham, G. H., '*Fungous diseases of fruit trees in New Zealand*', 1925, p. 137.

The conidiophores protrude through the stomata ; the spores may sometimes be formed in chains, hence the fungus is also known as *Alternaria Solani.* Under British conditions a species of Macrosporium, which may be identical with this, is a common saprophyte on damaged potato foliage, but there is no evidence of it being parasitic here.

M. Solani occasionally causes a black blotching of tomato foliage in Britain and produces brownish-black lesions on the fruits. Rosenbaum [21] states that a black foot rot of tomato plants is caused by it in the United States, where it is also partly responsible for ' damping-off ' of the seedlings.

Macrosporium tomato, Cke., causes the ' nailhead ' spot of tomato plants in the United States. This disease is characterized by the presence of circular, reddish-brown spots on the leaves, stems, and fruits.

Alternaria, Nees

Conidiophores fasciculate, simple or branched, dark-coloured ; conidia catenulate, clavate with long, narrow extremities, muriform, dark-coloured.

Some species are conidial stages of species of Pleospora.

Alternaria Brassicae, (Berk.) Bolle (= *A. herculea,* (E. and M.) Elliott)

This fungus causes a brown spotting of the leaves of turnips, kale, and kohlrabi in Britain, and a similar affection of other cruciferous crops in the United States, including a pod spot of seed cabbage and a discoloration of the ' heart ' of cauliflower. In Britain the fungus is of no economic importance. The conidia measure $90-350 \times 14-42\,\mu$. This fungus has been studied by Bolle [22] and Weimer [23].

The fungus known as *Alternaria circinans,* (B. and C.) Bolle, or *A. Brassicae,* (Berk.) Sacc., causes a somewhat similar spotting of cruciferous plants, but the spores of this species measure only $35-75 \times 9-17\,\mu$.

Alternaria Brassicae var. *nigrescens,* Pegl., causes a leaf spot of cucumbers grown under glass in England. In the United States it occurs on cucurbits out of doors.

Alternaria Citri, Pierce, causes a black rot of citrus fruits, especially navel oranges. Outwardly the fruit may appear little

affected, but the flesh is blackened. Infection occurs commonly at the stylar end, but Mr. R. G. Tomkins informs the writer that S. African oranges imported into Britain are often attacked at the stem end.

Alternaria Dianthi, Stevens and Hall, attacks the leaves and stems of some varieties of carnations, producing greyish spots, which become black with the conidiophores of the fungus. This species may also be the cause of a leaf spot and wilt disease of the carnation recently described by Corbett[24], in which infection may occur apparently through the soil as well as above ground.

Cercospora, Fries

Conidiophores simple or branched, dark-coloured; conidia vermiform and tapering, or nearly filiform, straight or slightly curved, multiseptate, sub-hyaline to dark-coloured.

Cercospora beticola, Sacc.

Conidia obclavate to needle-shaped, many-celled, nearly hyaline, $75-200 \times 3\cdot5-4\cdot5\,\mu$.

This fungus causes a leaf spot of mangolds, sugar-beet, and garden beet, and is sometimes very destructive on the Continent and in the United States. It occurs fairly commonly in Britain. When successive leaves have been killed by the fungus the crown of the root becomes abnormally elongated. The parasite also affects the fruits. It survives the winter either in old leaves in the form of sclerotia-like bodies, or upon the fruit. Pool and McKay[25, 26] point out that the germ-tubes of the spores enter the leaves only through the stomata, and that environmental conditions which cause the guard cells to open widely favour the disease. Mature leaves are most liable to attack, and it is such leaves which have the greatest number of stomata that open fully.

Cercospora Melonis, Cke.   Cucumber Leaf Blotch.

Conidia large, cylindrical, tapering, 7–9 septate, brownish, $80 \times 9\,\mu$.

Between 1896 and 1907 this fungus was very destructive to cucumbers grown under glass in England, but since then it has been rarely present owing to the introduction of more resistant varieties and to more careful plant sanitation. The disease first appears in the form of pale green, water-soaked

spots on the leaves, which turn brown. Affected leaves soon wither completely. The disease is only epidemic under conditions of high temperature and humidity.

Cercospora Apii, Fr.

This fungus is sometimes destructive to celery foliage on the Continent and in the United States, but it does not occur at present in Britain. Irregular greyish spots appear on the leaves, from which a greyish mould grows out, the conidia being almost hyaline.

Cercospora Violae, Sacc., attacks cultivated violets and violas. On the latter the blotches on the leaves are irregular in form ; at first they are purplish in colour, but become pallid. The conidia arise on the under surface.

Cercospora cantuariensis, Salm. and Worm., causes a leaf spot of the hop in England, which has been investigated by Wormald.* The spots are greyish and bordered by a purplish-brown line, which is surrounded by a yellowish zone. The spores are pale brown and very long, measuring $135 \cdot 508 \times 10-21 \mu$.

Cercospora Nicotianae, E. and E., causes a leaf spot of tobacco, which is sometimes troublesome in Rhodesia, Sumatra, and other countries.

Graphium, Corda

Conidiophores usually aggregated into dark-coloured coremia with head-like extremities ; conidia unicellular.

Graphium Ulmi, Schwarz   Dutch Elm Disease.

Coremia about 0·6 mm. high with black stalks and yellowish conidial heads which are often enveloped in mucilage ; conidiophores often verticillately branched ; conidia ovate or pear-shaped, unicellular, hyaline, $3 \cdot 4-1 \cdot 6 \mu$.

The conidia may bud in a yeast-like manner, and minute, spherical sclerotia sometimes occur.

A serious disease of elm trees began to appear in Holland about 1919, as described by Schwarz[26a], and it has since proved to be very destructive also in Germany, Belgium, and northern France. The disease has been attributed to various causes, including Graphium Ulmi and Micrococcus Ulmi, Brusoff. Although many aspects of the disease are still obscure, Wollenweber[27] in 1927 again isolated Graphium Ulmi from affected wood, which in pure culture reproduced the symptoms of the disease when inoculated into young elm trees.

* Wormald, H., 'The parasitism of the hop leaf-spot fungus Cercospora cantuariensis'. Trans. Brit. Myc. Soc., vol. 13, p. 32, 1928.

Trees of all ages, especially from 15–80 years, are liable to attack on diverse types of soil. The leaves of part of the crown become yellowish, wilt, and then turn brown. These symptoms extend rapidly over the whole tree, which dies back from above downwards. Dark brown streaks, often arranged in concentric zones, are found in the young wood of the trunk, branches, and twigs; the discoloration in the wood tends to spread from below upwards. The roots generally remain unaffected, but in young trees these also may be affected.

Several kinds of elm trees succumb to this disease, the most susceptible being *Ulmus montana*. So far *U. vegeta* has not been attacked, and, so far as is known, other kinds of trees are not affected by this disease.

Graphium Ulmi was isolated from a dead elm tree in England in 1927 by Wilson and Wilson [27a], who consider that the tree had been affected by the Dutch Elm Disease. During 1928 elm trees have been found to be attacked by this disease in several parts of England.

Fusarium, Link

Conidiophores simple to compoundly subverticillate, produced on a loose mycelium or over a layer of pseudoparenchyma. Conidia often of different kinds: macroconidia sickle-shaped, more or less pointed towards the apex, usually 3 or more septate, variously coloured in mass, but not grey or black; microconidia either of similar shape with few or no septa, or of a distinct type, oblong, ovate, &c., sometimes catenulate. The conidia may be produced singly, in small or large balls, in buttery layers (pseudopionnotes), in gelatinous layers (pionnotes), or in separate tubercular masses (sporodochia). Chlamydospores occur in some species, either within the macroconidia or in the hyphae. Sclerotia-like bodies are sometimes present.

Some species of Fusarium are now known to be conidial stages of Nectria, Gibberella, Hypomyces, &c.

Many species of the genus Fusarium are very difficult to identify because of their extreme variability. The taxonomy of the genus has been studied by Appel and Wollenweber [28], Wollenweber [29], Sherbakoff [30], Brown and Horne [31], Hansford [32], Brown [32a] and others.

Wollenweber [29] has divided the genus into a number of sections, and his classification is adopted here.

Section Elegans

All species have microconidia, mostly unicellular, scattered or in balls, ellipsoidal or reniform, averaging $5–12 \times 2–3.5\,\mu$; macroconidia sickle-shaped, mostly 3-septate, usually $25–40 \times 3–4.5\,\mu$, but also 4- and 5-septate and then usually $40–50 \times 3–4.5\,\mu$. Sporo-

dochia or pseudopionnotes often present. Conidia in mass mostly salmon-coloured. Chlamydospores terminal and intercalary, mostly unicellular, measuring 5–10 μ.

Many species are vascular parasites, causing wilts.

Fusarium conglutinans, Woll.   Cabbage Yellows.

Macroconidia few, no sporodochia or pseudopionnotes ; cultures on rice wine-red in colour.

This species causes a destructive disease of cabbage in certain parts of the United States, but it is unknown in Britain. When seedlings are infected the cotyledons wilt and the plants die. Later infections cause a yellowish, dwarfed type of growth, the lower leaves dropping off one by one. A cross-section of a cabbage stem affected by ' yellows ' shows discoloured vascular bundles. After death, pink masses of spores are formed on the surface of the stem. The fungus invades the plant through the roots, and once the soil has become infected the fungus may continue to live in it for some years, even though susceptible plants are not grown. The distribution of the fungus as a parasite is sharply limited by temperature. Tisdale [33] has shown that the fungus does not develop at soil temperatures lower than 63° F. (17° C.) and higher than 95° F. (35° C.). A low water-content of the soil also favours the disease.

The disease can only be effectively controlled by the use of resistant varieties, several of which have been selected by Jones and his co-workers [34]. Tims [35] states that although certain resistant varieties remain practically free from the disease at soil temperatures of 21–24° C. they are susceptible at temperatures of 27–33° C.

F. conglutinans var. *Callistephi* causes a serious wilt of the China Aster in the United States, as described by Jones and Riker [35a], and the same organism may be responsible for the wilt disease of this plant in England. Some varieties of China Aster are very resistant to the disease.

Fusarium bulbigenum, Cke. and Mass.

Sporodochia salmon-coloured ; macroconidia comparatively narrow, 3–5 septate, $40–50 \times 5–6 \mu$; chlamydospores colourless.

This Fusarium is often associated with the eelworm disease

of Narcissus bulbs, but it is probably capable of causing a rot without association with the eelworm. Massee[36] and Westerdijk[37] have investigated this disease, which is characterized by brown rings in the bulbs. Westerdijk[37] states that *F. gemmiperda* also causes a similar disease, this species being differentiated from *F. bulbigenum* by the chlamydospores being coloured blue in mass. If slightly diseased bulbs are planted, new bulbs which may be formed within them may also become infected, but the parent bulb often rots completely in the soil.

Fusarium oxysporum, Schl.

Macroconidia comparatively broad, almost entirely 3-septate, aggregated in a few, large sporodochia.

This species causes both a wilt of the potato plant and a dry rot of stored tubers in the United States. It sometimes attacks other plants, e.g. tomato and sweet potato there. It has not yet been recorded in Britain. The fungus enters the plant through the roots and grows chiefly in the xylem vessels, which become discoloured. The tubers are invaded through the stolons and show a brown discolouration of the vascular ring. If such tubers are planted, the plants developing from them frequently wilt. High temperatures are favourable for this fungus, the optimum being between 77° and 86° F. (25° and 30° C.). Chupp[38] states that another species, *F. eumartii*, causes a more virulent wilt of potatoes in the United States.

F. oxysporum, var. *Gladioli*, Massey, causes a corm rot of gladiolus, as described by Massey[38a]. The corms are affected in the field; the rot develops further in storage and may reduce the corms to brownish-black mummies. The lesions are slightly sunken, more or less circular, and frequently show concentric markings.

Fusarium zonatum (Sherb.), Woll., causes a bulb rot of onions in the field and in storage in the United States, as described by Link and Bailey[39]. This is the same fungus as that investigated by Walker and Tims[40] under the name of *F. cepae*, Hanzawa emend. Walker and Tims. Infection occurs from the soil, and in the field the leaves become yellow and die back from the tips; at the same time the roots turn pink and decay, and the bulbs become rotten. Walker and Tims[40] state that the disease is most serious

at soil temperatures of 28–32° C. In storage the rot develops most actively at temperatures of 20–30° C., but even at low temperatures (8–15° C.) much damage is done by premature sprouting of the bulbs, although there is little rot. Link and Bailey [39] have also found that *F. cepae*, Hanzawa emend. Link and Bailey is also destructive to onion bulbs. On the other hand *F. malli*, Taub., described by Taubenhaus and Mally [41] as causing a pink root rot of onions in Texas, proved to be non-pathogenic in the experiments of Link and Bailey [39].

Fusarium vasinfectum, Atk., causes a serious vascular wilt of cotton in the United States and Egypt. The macroconidia are chiefly 3-septate and are formed mainly in pseudopionnotes. The best hope of controlling the disease lies in the discovery of resistant strains of cotton.

Fusarium Lycopersici, Sacc.

Sporodochia numerous, often confluent, or pseudopionnotes often present, macroconidia almost entirely 3-septate.

In the United States this organism is chiefly responsible for tomato wilt, but in Britain it is less frequently the cause of this malady than is *Verticillium albo-atrum* (or *V. Dahliae*), chiefly because the optimum temperature for development, 29° C. (84° F.), is higher. The fungus enters the roots from the soil; the external symptoms of the wilt cannot be distinguished from those caused by Verticillium. The discolouration of the wood caused by the Fusarium is darker than that produced by Verticillium. Under greenhouse conditions in Britain the disease can be controlled by reducing the temperature, but, in countries where the temperatures are higher, control depends upon the use of resistant varieties or upon soil sterilization.

Fusarium cubense, Smith, emend. Brandes   Panama Disease of Bananas.

Conidia formed chiefly in sporodochia ; microconidia unicellular or uniseptate ; macroconidia mostly 3-septate, though also 4 or 5-septate.

Hansford [42] points out that there are numerous strains of this fungus differing slightly in their morphological features.

In recent years Panama Disease has devastated many thousands of acres of the Gros Michel variety of banana in Central America and Jamaica. It has also attacked this

variety in other parts of the world, including West Africa. Once land has become infected with this fungus, the Gros Michel variety, which is the most valuable commercial variety, can no longer be grown upon it.

The disease has been studied by Brandes [43], Hansford [42], and others. The first symptom is a yellowing of the outer leaf blades, which extends rapidly from the margin to the mid-rib. Such leaves begin to wilt almost immediately, and in a few days the leaf-stalks break close to the 'trunk' and hang down. Sooner or later the 'trunk' is blown over by the wind. Internally a diseased plant shows marked discolouration of the vascular bundles both in the rhizome and 'trunk'.

The fungus can grow independently in the soil, and infection may take place either through the wound at the base of the suckers used in propagation or through the roots. The fungus advances chiefly in the vessels, causing them to become discoloured. It is often spread by the use of slightly diseased suckers for planting. Spores are formed on the leaf-bases and leaf-stalks of diseased plants, and may be blown or washed away, infecting the soil in the vicinity.

Attempts have been made to check the spread of the disease in Jamaica by destroying all bananas within 22 yards of a diseased plant, and by preventing the subsequent growth of bananas upon the 'quarantined' area.

Other varieties of bananas, including the Canary banana, are very resistant to the disease, but these do not possess the commercial qualities of the Gros Michel variety. Until a resistant variety having the valuable qualities of the Gros Michel has been discovered, the disease is likely to spread disastrously.

Section *Martiella*

The conidial walls and septa are considerably thicker than in the Section *Elegans*; chlamydospores occur.

Fusarium caeruleum, (Lib.) Sacc.   Dry Rot of Potatoes.

Mycelium blue or bluish-green in mass; conidial masses brownish-white, pinkish, greenish or greenish-blue according to conditions; conidia chiefly 3-septate. $31-40 \times 4 \cdot 5 - 5 \cdot 5 \mu$; chlamydospores more or less spherical, bluish, $8-9 \mu$.

'Dry rot' of potatoes is most common in stored tubers of early varieties, particularly during the latter part of storage. In the earliest stage of attack the skin becomes somewhat sunken, and, as the disease spreads, the skin becomes folded into concentric wrinkles. The fungus grows deeply into the tissues and the whole tuber may become dry, hard, and greatly shrivelled. The flesh of a 'dry-rotted' tuber is brownish and often contains cavities filled with mycelium, which is frequently bluish in colour. Conidial pustules appear on the surface of the affected parts of the tuber; these are usually white on the exterior, but bluish-green within.

Pethybrige and Lafferty [44] have shown that infection may take place through scab spots, lenticels, eyes, and young sprouts, but it occurs most commonly through abrasions in the skin. The exact sources of infection are unknown, but *Fusarium caeruleum* probably exists in the soil. The rapid development of the disease during the later part of the storage life is doubtless bound up with chemical changes in the tubers. The early varieties 'Sharpe's Express', 'May Queen', and 'Duke of York' are particularly susceptible.

Tubers partly attacked by 'dry rot' should not be used as seed, as they frequently rot in the ground.

This species of Fusarium is almost exclusively the cause of 'dry rot' of potato tubers in Britain, but in the United States *F. trichothecioides*, Woll., and *F. oxysporum*, Schl., also often cause a very similar rot.

Section Discolor

For diagnosis see Wollenweber [29].

Fusarium culmorum, (W. G. Smith) Sacc.

Sporodochia or pionnotes ochraceous to salmon-coloured; conidia chiefly 5-septate, $30–45 \times 5\cdot5–7\,\mu$; chlamydospores intercalary, single, in chains or clusters.

In Britain, on the Continent, and in the United States *F. culmorum* and also *F. avenaceum* (Fr.), Sacc., cause a seedling blight, foot rot, 'deaf ears' and 'ear blight' in wheat and other cereals, as described by Atanasoff [45]. Bennett [45 a] has found that

these two fungi are the chief causes in the north of England of the diseases of cereals known as 'thinning out' and 'deaf ears'. The fungus may be carried over with the grain, or infection may occur through the soil. If the seedling is severely attacked, it may be killed outright, or the plant may be killed later by foot rot. In other cases the plant apparently grows normally until approaching maturity, when the ears ripen prematurely; these 'deaf ears' contain only shrivelled grains, if any. The roots and lower parts of the stems of these plants are permeated by mycelium. Such symptoms are sometimes mistaken for attack by *Ophiobolus graminis* ('Whiteheads'). Lastly, in wet weather the fungus may attack the ears after flowering, covering them with a pink, mucous mould ('ear blight'), which may injure the grain seriously, sometimes killing the embryo.
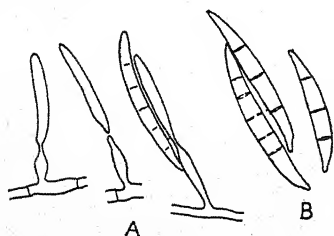
FIG. 62. *Fusarium herbarum*: (A) formation of conidia, × 350; (B) conidia, × 600. (*W. J. Dowson.*)

Where this disease is prevalent the grain should be treated with a fungicide before sowing.

F. culmorum also causes considerable havoc among carnations under glass in England, especially where grown in soil beds. This disease is known as 'Carnation Wilt'. A similar disease has been described by Delacroix and Maublanc [46] in France. The fungus usually enters the plant from the soil, chiefly through wounds, passes into the vessels of the roots and lower part of the stem, and causes a wilt and gradual desiccation of the shoots. At first only a single branch may be affected, but the entire plant is killed in time. The fungus may also infect the cut extremities of the shoots. The disease is most severe where carnations are grown in the same soil year after year, the soil often becoming 'sick'. Extreme wetness of soil and a relatively high temperature favour the disease. Soil beds in greenhouses should be steam sterilized at intervals, or, alternatively, other plants should be grown in rotation. Cuttings should be taken only from healthy plants. There

are considerable varietal differences in susceptibility to this wilt, and in time probably only the more resistant types will be cultivated. Mr. W. J. Dowson, who has investigated Carnation Wilt in England, informs the author that another disease of carnations under glass, which leads to a die-back of pinched-out shoots, is caused both by *F. culmorum* and *F. herbarum*, (Corda) Fr.

Section Lateritium

For diagnosis see Wollenweber[29].

Fusarium fructigenum, Fr.

Pustules pinkish ; conidia mostly 5-septate ; chlamydospores rare ; bluish green sclerotia are formed on culture media.

Kidd and Beaumont[47] have described this species as the cause of a rot of apples and pears in storage in Britain, infection frequently occurring through the ' eye '. The same species causes a bud rot of certain varieties of apples in some parts of England. It is not yet known how infection of the buds is brought about.

Section Ventricosum

For diagnosis see Wollenweber[29].

Fusarium Lini, Boll.   Flax Wilt.

Conidial pustules not densely aggregated, cream to flesh-coloured ; conidia 3-septate, ventricose ; chlamydospores terminal.

This species causes a serious disease of flax in certain parts of the United States, but it is of less importance in the flax-growing districts of Northern Ireland, probably because of lower soil temperatures there. Flax-plants may be infected from the soil from the seedling stage onwards : young plants may be killed outright, but older plants often become yellowish before they wilt and die. The fungus survives in the soil for several years, especially on the decaying remains of flax plants ; it is probably an important factor in rendering the soil ' flax-sick ' where the crop has been grown at short intervals.

In recent years wilt-resistant varieties have been introduced into cultivation in the United States, and in consequence the

disease is not so serious as formerly. Tisdale [18] states that
when the fungus begins to attack the roots of resistant
varieties it is prevented from entering the vascular system by
the suberization of the cell walls or by the formation of a cork
layer in the cortex.

Broadfoot and Stakman [49] have distinguished several forms
of this fungus which differ greatly in virulence.

Meria, Vuillemin

Conidiophores emerging through the stomata of the host, each
segment bearing a single hyaline, unicellular conidium formed at
the end of a short sterigma. The systematic position of this
genus is uncertain.

Meria Laricis, Vuillemin, attacks the needles of the larch, especially
in nurseries, causing them to turn yellow and then brown. The
disease sometimes spreads epidemically. Infection occurs in the
spring from conidia formed on leaves which have lain on the
ground during the winter. The conidia measure $8-10 \times 2 \cdot 6 \ \mu$.

MYCELIA STERILIA

This is a heterogeneous assemblage of fungi in which no spore
forms are yet known. Sclerotia are produced by some species.

Rhizoctonia Crocorum, (Pers.) DC.   Violet Root Rot.

This omnivorous fungus attacks a great variety of plants,
including the underground parts of Crocus, lucerne, clover,
seakale, Asparagus, carrot, sugar-beet, young trees, &c. It
may also cause a rot of mangolds and potatoes in the field
or in storage. The violet or purplish coloured mycelium is very
characteristic. When growing plants are affected by it the
mycelium may spread from plant to plant. The fungus kills
the plant tissues as it grows in them. Small reddish-violet
sclerotia are formed by the mycelium; these serve as a means
of tiding the fungus over adverse conditions, and give rise to
mycelium again on germination. Bodies somewhat resembling
the sclerotia are formed where the mycelium from the soil
comes into contact with, and penetrates, the host, and it is by
way of these 'infection cushions' or 'corps miliaires' that
nutriment is transmitted from the host to the mycelium
outside it.

Buddin and Wakefield [50] have brought forward evidence that *Helicobasidium purpureum*, (Tul.) Pat., one of the Auriculariales, is the sporiferous stage of *R. Crocorum*. Pure cultures of both are essentially the same, and certain strains of each produce conidia of the Tuberculina type in culture.

Sclerotium Tuliparum, Kleb. (= *Rhizoctonia Tuliparum*, (Kleb.) Whetzel and Arthur) Grey Bulb Rot.

This disease of tulip and other bulbs was first diagnosed by Klebahn [51, 52] in Germany. It occurs extensively in Holland, in the United States, where it has been investigated by Whetzel and Arthur [53], and in Britain. Klebahn points out that the fungus also attacks *Iris hispanica*, hyacinths, *Fritillaria imperialis*, yellow Narcissus and *Scilla siberica*, and in Britain it is sometimes destructive to *Iris reticulata*, as recorded by Brooks [53 a].

The first indication of the disease in a bed of bulbs is the presence of gaps where the plants have failed to come up, or more rarely the occurrence of malformed shoots which do not flower. Entire beds of bulbs may be thus affected. When the diseased bulbs are dug up they are found to be more or less completely rotten. Infection occurs from the soil, the first part to be attacked being the young shoot growing up from the bulb. The mycelium passes into the shoot and thence into the bulb. The greyish or brown sclerotia frequently adhere to the young shoots and are considerably mixed with soil. The greyish mycelium of the fungus often extends over the surface of the rotten bulb. The sclerotia are easily detached from the diseased bulbs and may remain in the soil in a dormant condition for some time. Bulbs planted in contaminated soil are liable to infection by the mycelium which arises from the sclerotia. The sclerotia are almost black when dry; they are roughly spherical in shape and are about 3–6 mm. in diameter. The sclerotia in section are seen to have a very thin rind, which is ill-defined, the interior consisting of a pseudo-parenchyma of thin-walled, spherical cells. No sporing stage is known in the life-history of the fungus.

The disease is doubtless chiefly disseminated by the planting

of slightly diseased bulbs, particularly of kinds which are not so badly attacked as are tulips. Once soil has become contaminated, a long interval should elapse before bulbs are again planted in it. Whetzel and Arthur [53] state that the sclerotia can be destroyed by steam-sterilizing the soil or by treating it with formalin (1 in 50) at the rate of 1 pound of formalin to 6 square feet of soil.

Sclerotium Rolfsii, Sacc.

The sclerotia of this fungus are brown and small, about the size of a mustard seed. The mycelium grows in the form of white strands or fan-shaped expansions; clamp connexions occur at the septa. In warm countries, especially in the southern part of the United States, this fungus is a virulent parasite of many crop plants, e. g. beans, maize, potatoes, tomatoes, cabbage, rhubarb, &c., attacking them from the soil near ground level. Uninjured tissues are readily invaded by the mycelium. When young plants are attacked they quickly die; the fungus subsequently appears at the base of the stem as a white mass in which the sclerotia are embedded. The latter may retain their vitality in the soil for long periods, ultimately germinating by the formation of mycelial strands. The disease usually appears at a few centres in a field, from which it spreads centrifugally. When older plants are attacked the shoots turn yellow before they wilt and die.

According to Taubenhaus [54] there are no distinct physiological strains of the fungus, the mycelium from one host being able to attack any other host. The parasite is most destructive in light sandy soils well provided with moisture. If buried deeply the scelerotia cannot germinate.

This fungus has been found occasionally in England on imported corms of the Arum Lily.

According to investigations made by Nakata [54a] in Japan *Hypochnus centrifugus*, (Lév.) Tul. (= *Corticium centrifugum* (Lév.) Bres.), one of the Thelephoraceae, is the sporing stage of *Sclerotium Rolfsii*.

Sclerotium Oryzae, Catt., kills young rice plants and greatly reduces the yield of mature plants in Italy, India, Malaya, and

Japan. Butler [55] states that a characteristic feature of the disease in older plants is the putting forth of green shoots from the base when the rest of the crop is ripening. On splitting open the stem of such plants a mycelium is found near the base, which bears small, round, black sclerotia. These persist in the soil, and the mycelium arising from them attacks uninjured tissues.

Sclerotium Gladioli, Massey.* Dry Rot of Gladiolus.

This serious disease of Gladiolus and Montbretia corms has been described by Drayton [56] in Canada. It occurs also in Holland and England. So far as is known at present, the causative fungus possesses no functional spores, reproduction being effected by minute, black sclerotia, $\frac{1}{10} - \frac{1}{5}$ mm. across. The fungus is referred therefore to the form genus Sclerotium. On harvesting diseased Gladiolus corms the soil adheres to them frequently in lumps, and the scales are found to be dull brown or spotted, and partially decayed. The surface of the corm bears small, dry, sunken lesions, reddish brown to black in colour, which may be confluent. The lesions may be shallow, and are then separated from the healthy tissue by a cork cambium, but in severely affected corms the vascular bundles are penetrated and discoloured by the fungus. The disease sometimes progresses rapidly in storage, and, by the spring, many corms may be dead.

In the growing plant the first sign of the disease is yellowing and then wilting and browning of the outer leaves.† The stem decays at soil level and the leaves fall over. Numerous sclerotia are found at the base of the leaf sheaths. The roots of a diseased plant are poorly developed and more or less decayed.

Mr. F. L. Drayton informs the author that if diseased corms are grown in uncontaminated soil about 25 per cent. of the plants become diseased, but that if healthy corms are grown in contaminated soil 70 per cent. of the plants are affected. He states that infection invariably occurs through the soil. In a corm planted in a diseased condition the fungus grows out from the lesions into the soil and then attacks the base of the outermost leaf; at the same time the newly formed contractile roots are invaded by the fungus, and it is due to the destruction of these that the plant turns yellow and wilts.

As the fungus persists in the soil for at least four years, land contaminated with it should not be used for growing Gladiolus. Only healthy corms should be planted; this is best ensured by removing the scales just before planting and discarding any corms which show the lesions of the disease.

* See Massey, L. M., 'Dry rot of Gladiolus corms'. *Phytopath.*, vol. 18, p. 519, 1928.

† In the destructive leaf and corm disease of Gladiolus caused by *Bacterium marginatum*, which has been described by McCulloch [57], the characteristic symptoms in the field are yellowing, wilting, and rotting of the central leaves before the outer leaves are seriously affected.

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DISEASES CAUSED BY GREEN ALGAE

THE great majority of the Green Algae (Chlorophyceae) are lowly organized plants containing chlorophyll, which live in fresh water. Reproduction is often effected by zoospores, or by various kinds of resting spores, which may be the result of sexual fusion. A few of these algae are parasitic or semi-parasitic in the tissues of the higher plants : these are orange or yellowish-green in colour, as true chlorophyll is not formed in them.

Cephaleuros parasiticus, Karsten   Red Rust of Tea.

There is some uncertainty about the identity of the species of Cephaleuros which causes a serious disease of tea in northern India and Ceylon. According to Petch[1], *C. parasiticus* is parasitic on both stems and leaves and *C. mycoidea* is chiefly epiphytic on the leaves. The last-named species occurs commonly as a surface growth on the leaves of many tropical plants ; the tissues below the alga may be killed, but are not usually penetrated by it. *C. parasiticus* produces purplish-red or blackish spots on the leaves : it grows chiefly within the tissues, the filaments being confined to the air-spaces between the cells. At maturity, reddish, hair-like branches of the alga protrude from the spots and terminate in large, spherical cells (sporangia). Zoospores, formed in these sporangia, are splashed about by rain, and infect other leaves. Much more serious, however, is the attack of this alga on the stems. Both young and old stems may be penetrated by the algal filaments, the effect being to weaken the bush greatly. After a time reddish, hair-like filaments (' Red Rust ') grow out from the surface and form reproductive bodies, as on the leaves. The chief outbreak of ' Red Rust ' occurs after pruning.

' Red Rust ' is essentially a disease of weak bushes, for if vigorous stems are invaded by the alga, the host checks the spread of the parasite by the formation of cork barriers. Weak stems, however, do not react in this way, so that the alga spreads extensively. Treatment for this disease consists in effecting

improvements in cultivation, manuring, and pruning, which lead to the development of vigorous bushes. In the reproductive stage the alga cannot be controlled by spraying as the hair-like outgrowths are not easily wetted, but spraying with Bordeaux mixture or with lime-sulphur immediately after pruning is often attended with good results.

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CHAPTER XXII

FUNGICIDES

As there are several books on Fungicides already available, this subject will not be treated in detail in the present chapter. Those who desire fuller information about these substances and their modes of preparation and application should consult the following books:

E. Bourcourt. *Insecticides, Fungicides and Weedkillers.* Translated. Scott, Greenwood & Son, 1913.

E. G. Lodeman. *The Spraying of Plants.* The Macmillan Co.

M. Hollrung. *Die Mittel zur Bekämpfung der Pflanzenkrankheiten.* Paul Parey, Berlin, 2nd edition, 1914.

O. G. Anderson and F. C. Roth. *Insecticides and Fungicides, Spraying and Dusting Equipment.* Willis, New York, 1923.

P. J. Fryer. *Successful Spraying and how to achieve it.* Benn, 1923.

E. Vogt. *Die chemischen Pflanzenschutzmittel, ihre Anwendung und Wirkung.* De Gruyter Co., Berlin and Leipzig, 1926.

W. Trappmann. *Schädlingsbekämpfung.* S. Hirzel, Leipzig, 1927.

The Duke of Bedford and S. U. Pickering. *Science and Fruit-growing.* The Macmillan Co, 1919.

Fungicides may be divided into two classes: (*a*) those which are immediately effective when applied to a growing fungus, as, e. g., carbolic acid, sulphuric acid, washing soda; and (*b*) those which are essentially protective and which should be applied therefore before the fungus has established itself on the host, as, e. g., Bordeaux and Burgundy mixtures. Sulphur, lime-sulphur, and other sulphur compounds belong to an intermediate category, although it is often best to apply them as protective fungicides. Protective spray fluids should be applied in the form of a fine mist, so that on drying, a thin film of the fungicide covers the whole of the shoots. If the fungicide is applied as a dust or powder it should be extremely finely divided and should be applied, if possible, when the dew is on the leaves. Fungicides should be innocuous to the host plant, and to ensure safety in this respect, particular care in preparation and application is required.

The fungicidal action of many of these bodies comes into play by the gradual formation of toxic substances. The mode of action of Bordeaux mixture has been investigated by Pickering [1] and by Barker and Gimingham [2]. The fungicidal properties of certain sulphur compounds have been investigated by Eyre and Salmon [3], Eyre, Salmon, and Wormald [4], Horton and Salmon [5], and Goodwin, Martin, and Salmon [6], and the fungicidal properties of 'wettable sulphur' have been studied by Goodwin and Salmon [7]. Little is yet known about the exact manner in which sulphur and its compounds act as fungicides.

Bordeaux Mixture

This is a mixture of basic compounds of copper formed by the interaction of copper sulphate and lime in water. It is particularly effective in preventing attack by 'downy mildews'. Many formulae are employed for making this fungicide, the following being one of the most generally useful:

Copper sulphate	4 lb.
Lime	4 lb.
Water	50 galls.

Method of making:

Dissolve overnight 4 lb. of copper sulphate in 45 galls. of water in a barrel. Slake gradually 4 lb. of stone ('fat') lime, and make up the milk of lime to 5 galls. Strain the milk of lime through a fine sieve into the solution of copper sulphate, and stir vigorously, when a flocculent blue precipitate will be formed. Transfer to the spraying machine, which should be made of, or lined with, copper, and which should be provided with nozzles throwing a fine, mist-like spray.

Alternatively, 5 galls. of concentrated copper sulphate can be added to 45 galls. of strained, diluted milk of lime.

Bordeaux mixture as above should not be used for spraying certain sensitive varieties of fruit trees after blossoming, as it may cause scorching of the foliage and russetting of the fruit. For spraying such fruit trees after flowering and for spraying other delicate plants 'excess-lime' Bordeaux mixture, made according to the following formula, should be used:

Copper sulphate	.	.	.	.	3 lb.
Lime .	.	.	.	.	.	10 lb.
Water .	.	.	.	.	.	50 galls.

Even this Bordeaux mixture may cause slight russetting of the fruit of some varieties, and it should not be used for spraying the apple 'Cox's Orange Pippin' and a few other tender varieties after blossoming.

For controlling leaf-eating insects at the same time, lead arsenate paste may be incorporated at the rate of 2 lb. per 50 gallons of Bordeaux mixture. Nicotine also may be mixed with Bordeaux mixture (one part of nicotine, 95–98 per cent. pure, in 2,000 parts of B. m.) in order to destroy leaf-sucking insects (aphides, &c.) simultaneously.

Where there is difficulty in obtaining water, Bordeaux mixture in powder form is sometimes used for protecting potatoes against Blight, but it is not so efficacious as the wet spray.

In North America a Bordeaux mixture dust containing lead arsenate is used for controlling Apple Scab. This powder consists of 10 parts copper sulphate, 10 parts lead arsenate, and 80 parts lime.

For spraying citrus trees 1 per cent. paraffin emulsion is usually added to Bordeaux mixture in order to avoid injury to fungi which parasitize scale insects. Paraffin emulsion is made by dissolving 2 lb. of soft soap in 1 gallon of hot water, and then gradually adding 2 gallons of paraffin, the mixture being churned vigorously as the paraffin is added. The emulsion should be added slowly to freshly prepared Bordeaux mixture while the latter is being agitated.

In Kenya, Dowson [8] has successfully used calcium carbide instead of lime for making a modified Bordeaux mixture, $1\frac{1}{2}$ lb. of carbide being used, with 4 lb. copper sulphate, per 40–50 gallons of spray fluid.

Goodwin and Salmon [7] and Goodwin, Salmon, and Ware [7a] have shown that hydrate of lime can be used instead of quicklime for making Bordeaux mixture, 6 lb. of hydrate of lime being equivalent to 4 lb. of quicklime. Hydrate of lime should

be of 95 per cent. purity and should be kept in paper-lined bags as it changes to carbonate of lime on exposure to the air.

Burgundy Mixture

This is made like Bordeaux mixture, but washing soda replaces lime. The formula is:

Copper sulphate	.	.	.	.	4 lb.
Washing soda .	.	.	.	.	5 lb.
Water	.	.	.	.	.	50 galls.

Where lime of good quality or hydrate of lime cannot be obtained Burgundy mixture should be used instead of Bordeaux mixture. Burgundy mixture is very adhesive, but it is rather more liable than Bordeaux mixture to scorch foliage, and for this reason it should not be used to spray fruit trees in leaf.

Ammoniacal Copper Carbonate

For very delicate foliage (e.g. peach) and where it is undesirable to stain the fruit, this fungicide should replace Bordeaux mixture. The formula is:

Copper carbonate	.	.	.	.	5 oz.
Strong ammonia	.	.	.	.	3 pints.
Water	.	.	.	.	.	50 galls.

Method of making:

The copper carbonate should be made into a thin paste with water, and then one pint of the ammonia diluted to one gallon with water should be added; the mixture should be shaken vigorously and allowed to settle, when the blue fluid is poured off; further quantities of diluted ammonia are then added to the residue until the whole has been dissolved; the remainder of the water should then be added. This fungicide should be used immediately as it does not keep well.

Sulphur

This is one of the best fungicides for mildews (Erysiphaceae). Flowers of sulphur or very finely ground sulphur should be used, and they are best applied when the dew is on the leaves. For controlling mildews in greenhouses sulphur is often vaporized.

In North America Apple Scab has been prevented by dusting

with a mixture consisting of 90 per cent. finely divided sulphur and 10 per cent. powdered lead arsenate.

American Gooseberry Mildew has been controlled in England by dusting the gooseberry bushes with flowers of sulphur or with ground sulphur just before the blossoms open.

'Wettable' Sulphur

Finely divided sulphur cannot be mixed with water, but if moistened with an alcoholic solution of oleic acid or with a dilute solution of soft soap, the sulphur becomes wettable. Goodwin and Salmon [7] point out that a thick suspension of sulphur, previously wetted with oleic acid, in a 1 per cent. solution of soft soap is more toxic to *Sphaerotheca Humuli* on hop leaves than either sublimed or finely ground sulphur. Nicotine can be incorporated with 'wettable' sulphur.

Lime-sulphur (Calcium polysulphides)

This is made by boiling together lime and sulphur. Reliable commercial brands of lime-sulphur are now available, of specific gravity 1·3. For use, the concentrated solution is diluted to a specific gravity of 1·01 (1 to 29), 1·005 (1 to 59), or 1·003 (1 to 99).

For the control of Apple Scab the trees should be sprayed in the pink blossom bud stage with the fluid of specific gravity 1·01, and subsequently with that of specific gravity 1·005, or 1·003 for the more delicate varieties. Lime-sulphur of specific gravity 1·01 applied after flowering, causes some fall of fruit, as indicated by Grubb [9], especially if the trees were not sprayed with this substance before the blossoms opened.

If desired, lead arsenate or nicotine can be incorporated with lime-sulphur, as described under the heading of Bordeaux mixture. With lime-sulphur + lead arsenate the film is deposited more evenly if a 'spreader', e. g. calcium caseinate, is added at the rate of 1 lb. per 100 gallons.

Ammonium Polysulphide

This fluid was introduced by Eyre, Salmon, and Wormald [10, 11] for late application to gooseberry bushes attacked by American mildew, as it does not spot the fruit. With the '1919' ammo-

nium polysulphide half a gallon should be diluted to 100 gallons by the addition of water, and a 'spreader', e.g. saponin, should be added at the rate of 2 oz. per 100 gallons of spray fluid. It is sometimes advantageous to incorporate 5 lb. of soft soap with 100 gallons of this fungicide.

Liver of Sulphur (*Potassium polysulphides*)

This fungicide is useful for controlling mildews, but lime-sulphur has almost entirely replaced it. Liver of sulphur is used at a strength of 4 lbs. per 100 gallons of water. It is generally advisable to add a 'spreader', e. g. saponin (as above) or flour (5 lb. per 100 gallons), the latter being first made into a paste.

Washing Soda

Washing soda, together with soft soap, as in the following formula, may be used for controlling American Gooseberry Mildew, but, according to Nattrass [12], Burgundy mixture is better for this purpose:

Washing soda	.	.	.	.	.	9 lb.
Soft soap	.	.	.	.	.	5 lb.
Water	.	.	.	.	.	50 galls.

Carbolic Acid

A weak solution of carbolic acid (one desertspoonful in a gallon of water) containing soft soap (2 oz. per gallon) is useful for controlling Rose Mildew.

Tar-oils

The practice of spraying fruit trees in winter with a tar-oil (tar-distillate) wash for the control of aphides and other insects by killing their eggs has now become general in England, and there is some indication that this practice may reduce 'Brown Rot' in plums. For spraying plum trees the tar-oil should be used at a strength of 6 per cent.; it should be applied in December or early in January.

Cheshunt Compound

This was introduced by Bewley [13] at the Cheshunt Experimental Station for the control of certain 'damping-off fungi'.

The mixture consists of 2 parts by weight of copper sulphate and 11 parts of ammonium carbonate. Fresh ammonium carbonate is ground to a fine powder and is then thoroughly mixed with powdered copper sulphate and stored in a stoppered bottle for twenty-four hours before use. The solution is prepared by dissolving 1 oz. of the dry mixture in a little hot water, which is then added to 2 gallons of water. Plants can be watered with this solution without injury, but 'damping-off' fungi in the soil are killed by it.

Sulphuric Acid

This is used for destroying parasitic fungi in the soil of nursery beds, especially those of forest trees. For each square foot of the nursery bed one-third of a fluid oz. of commercial sulphuric acid should be diluted with $1\frac{1}{2}$ pints of water. The beds should be watered with the solution immediately after the seed has been sown; subsequently, ordinary water should be applied during germination in order to prevent undue concentration of the acid.

Soil Sterilization by Heat

For modern methods of sterilizing greenhouse soils by heat the reader should consult the account given by Bewley [14].

Formalin, Copper Sulphate, and Copper Carbonate

For prevention of seed-borne diseases by means of these substances, see pages 214, 223, etc.

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INDEX

(An asterisk denotes an illustration.)